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The Effects of Reverberant Blast Trauma (Impulse Noise) on Hearing: Parametric Studies

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13. ABSTRACT *(Maximum 200 words)*
The effects of exposure to high levels of reverberant blast waves (impulse noise) on the auditory system using an animal (chinchilla) model are reported. Blast waves were generated by two different shock tubes which produced wave signatures having different spectral distributions of energy. Intensities for each blast wave source were 150, 155, and 160 dB peak SPL with exposure to 1, 10, or 100 impulses. Repetition rates were fixed at one impulse/sec. There were 15 subjects per group. Brainstem evoked potentials were used to estimate temporary and permanent threshold shifts and conventional surface preparations of the cochlea were used to quantitatively assess sensory cell loss. In addition, pre- and postexposure cubic distortion product otoacoustic emissions (3DPE) were collected on a subset of these animals for correlation with audiometric and histological indices of trauma. Trauma systematically increased with sound exposure level (SEL) and was dependent on the energy spectrum of the impulse. An application of the A- and P-weighting functions to the spectra improved the correlations between indices of trauma and SEL. The 3DPE data were consistent with PTS and, overall, both measures were correlated with sensory cell loss.

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FOREWORD

Disclaimer:

Opinions, interpretations, conclusions and recommendations are those of the authors and are not necessarily endorsed by the Department of the Army. Citations of commercial organizations and trade names in this report do not constitute an official Department of the Army endorsement or approval of the products or services of these organizations.

Animal Use:

In conducting the research described in this report, the investigators adhered to the "Guide for the Care and Use of Laboratory Animals," prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council (DHHS Publication No. (NIH) 86-23, revised 1985).

Roger P. Hamernik, Ph.D.

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I. INTRODUCTION

There are a number of different standards suggested for exposure to impulse/impact noise (e.g. Coles, et al., 1968; OSHA, 1974; Smoorenburg, 1982; Pfander et al., 1980; and NAS-CHABA, 1992). Although each of these criteria has its proponents, none of them are in complete agreement with the existing empirical data (Smoorenburg, 1990). Unfortunately, there is an extremely limited empirical data base upon which a new standard can be built. What is needed is a criterion based upon a cohesive, systematically-acquired body of experimental data. The need for such a data base has been emphasized by, for example, von Gierke (1978, 1983), Ward (1983), the NATO Study Group RSG.6 (1987), and the U.S. National Academy of Sciences CHABA Working Group (1992). The difficulties associated with generating such a data base are compounded by the extremely broad range of high intensity noise transients that exist in various industrial and military environments. For example, in industry, reverberant impacts with variable peak intensities usually under approximately 140 dB, often occur at very irregular intervals. At the other extreme, the diversity of military weapon systems produce impulses which originate as the result of a process of shock wave formation and propagation following an explosive release of energy. These waves, which can have peak levels well in excess of 180 dB, can be either reverberant or non-reverberant in nature depending upon the environment in which they are encountered. Trying to develop a single standard to cover this large dynamic range of "acoustic" signals is a formidable task.

II. BACKGROUND

Recent critiques and analyses of impulse noise exposure criteria (Smoorenburg 1990, 1982; NATO, 1987; NAS-CHABA, 1992) stress the paucity of experimental data from which these various criteria evolved. In particular, two areas of the existing data base that have been singled out as being deficient are those relating to the effects of high level reverberant impulse noise exposure and the effects of the impulse energy spectrum. While data on spectral effects have recently begun to accumulate (see e.g., Price 1979, 1983, 1986; Patterson et al., 1986, 1990(a,b); Hamernik et al., 1990), there is virtually no information available on reverberant blast wave exposure. Reverberant stimuli are typically encountered by military personnel when a weapon is discharged in, or a projectile impacts on, a confined environment (e.g., enclosed urban area or armored vehicle). Stimuli of this type comprise some of the most hazardous exposures for the auditory system that are to be found in military environments (Walden et al., 1971, 1975) and represent a type of stimulus for which virtually no experimental data base exists from which one can estimate the expected level of auditory system trauma (primary blast wave injury). This report presents the results of a series of experiments that were designed to explore the relation between hearing trauma and exposure to spectrally different, high-level reverberant blast waves.

A literature review on the auditory effects of reverberant blast waves has not uncovered any systematically acquired data which will contribute to furthering our understanding of this problem. However, several laboratory and epidemiologic studies indicate the potential severity and complexity of the problem. For example, Hynson et al. (1976) showed that a free field impulse which is followed by reflected components can contribute disproportionately to the eventual permanent

threshold shift (PTS) and cochlear lesions. This study, however, was based upon a small number of animals (two groups with five animals per group) and has not been repeated. More recently Roberto et al. (1989) reported on the anatomical changes in pigs and sheep exposed to high-level reverberant blast waves within an armored vehicle. Although no measurements of hearing were made, the lesions in the cochlea and middle ear were extensive and probably the result of direct blast wave-induced, mechanical damage to the auditory system (Luz and Hodge, 1971). The levels, while extremely high (approximately 195 dB), were typical for the type of projectile impact and nature of the armored vehicle. Although not a study of hearing, Clemedson and Jonsson (1976) indicated that exposure to reverberant blast waves are more hazardous to the respiratory system than are exposures to the same type of stimulus in a non-reverberant system. Since the total energy of the exposure is increased and under certain circumstances peak levels are also increased, this result, showing an exacerbation of effect in a reverberant system is not surprising and will most likely also be true for the hearing end organ. Demographic data such as that of Walden et al. (1971, 1975) can be interpreted as indicative of an increased risk of hearing loss when personnel are exposed to high noise levels in acoustically hard (reverberant) environments. Additional, but much more circumstantial, evidence emphasizing the potential for trauma from reverberant impulses can be found in clinical reports, such as Salmivalli (1967) or Smyth (1974). These reports document the severe hearing loss following acute acoustic trauma from a variety of military and non-military sources. While there is little or no documentation of the acoustic signal, one can assume with some confidence from the circumstances of the trauma, that the signal was reverberant.

The energy spectrum of an impulse is also widely acknowledged to be important in risk assessment, although relatively little experimental data is available that can be used to understand the role of spectrum in the production of hearing loss. In fact, one of the surprising features of the existing or proposed impulse noise exposure criteria is the general lack of specific consideration that is given to the frequency-domain representation of the impulse, a point frequently raised by Price (1979) and others. Some deference is, however, given to the spectrum in these criteria, but in an indirect way [e.g., through the use of A-weighting of the stimulus or through the handling of the A and B duration variables; see Coles et al. (1968)]. A more direct spectral approach to the evaluation of impulses and impacts was proposed by Kryter (1970). His suggestions, while based upon sound reasoning, never caught on. The Kryter approach appeared attractive in its ability to predict the amount of temporary threshold shift measured two minutes after exposure (TTS_2) to a noise transient, provided that the TTS_2 was not very large or, alternatively, that the levels of the transient in any given frequency band were not excessive. Price (1979, 1983, 1986), to some extent has tried to build upon and extend the Kryter approach by considering the spectral transmission characteristics of the peripheral auditory system. Price's reasoning led to the following conclusions: (1) There is a species specific frequency, f_o, at which the cochlea is most vulnerable and that impulses whose spectrum peaks at f_o will be most damaging. This would appear to be true, according to Price, regardless of the distribution of energy above and below f_o. For humans the suggested frequency is 3.0 kHz. (2) Relative to the threshold for damage at f_o, the threshold for damage should rise at 6 dB/octave for $f_p<f_o$ and at 18 dB/octave for $f_p>f_o$, where f_p = spectral peak of the impulse. Thus, a model for permanent damage was developed which is

amenable to experimental testing. In subsequent studies, Price (1983, 1986) attempted to relate, with varying degrees of success, experimental data obtained from the cat to the predictions of his model. The data reported by Price are limited, and suffer from a large variability which, because of the small number of subjects in the various exposure groups, makes general conclusions tentative. More recently, Hamernik et al. (1990), Patterson and Hamernik (1990) and Patterson et al. (1993) have reported on an extensive series of parametric studies in which the spectra of non-reverberant impulses were varied. A review of the literature indicates that, except for the studies mentioned above, there are few, if any, other published results obtained from experiments specifically designed to study the effects of the spectrum of an impulse on hearing trauma.

The Patterson and Hamernik (1990), Hamernik et al. (1990), and Patterson et al. (1993) reports on the results of exposures to several types of impulse noise and blast waves represent one of the most extensive compilations of data on spectral effects that exist. These studies have shown that it is possible to bring order to relations among permanent threshold shifts produced by exposure to impulsive noise and the spectrally weighted energy of the exposure. What was encouraging about these data was that an empirical weighting function obtained using impulses that were generated by conventional electro-acoustic methods could be used to unify the results obtained using high-level shock tube generated blast waves. These results were also in general qualitative agreement with the predictions of Price.

Our experimental approach in the series of reverberant exposures reported here is one that relates the parameters of an exposure (peak levels, energy, frequency content, number of impulses, etc.) with measures of hearing threshold recovery, permanent threshold shifts, and quantitative measures of cochlear sensory cell loss (cochleograms). All measures of auditory threshold were obtained from auditory evoked potential audiometry using chronically-implanted electrodes. This physiological measure correlates well with psychoacoustic measures of threshold. Thus, we have two very different measures of trauma, the hearing threshold measures and the anatomical cell loss. In addition to these measures of trauma, cubic distortion product emissions were collected on a subset of the experimental group in order to initiate an effort to establish correlations among the otoacoustic emission data and audiometric and histological indices of trauma.

III. METHODS

Additional details of the methodology are available in Hamernik et al. (contractor reports) ADA 203-854, ADA 206-180 and ADA 221-731. Briefly, the basic experimental protocol that was common to all of the experiments reported here consists of the following steps: (1) Preexposure audiograms were measured on each animal. (2) Each animal was exposed to calibrated blast waves. The temporal and spectral characteristics of the noise were recorded. (3) The animal's evoked response thresholds were again measured immediately after exposure and at regular intervals up to 30 days after exposure. At 30 days postexposure, the audiogram was again measured to establish the animal's permanent threshold shift (PTS). (4) Each animal was euthanized and its cochlea was prepared for microscopic analysis. Cochleograms, which provide a quantitative description of the extent and location of the hair cell (sensory cell) lesions, were prepared for each cochlea. In

addition to requiring normal preexposure audiograms, each animal was screened using tympanometry in order to determine the status of the middle ear before and after exposure. Cubic distortion product otoacoustic emissions (3DPE) also were measured on some animals when the appropriate equipment became available, as explained below.

A. Subjects: The chinchilla was used as the experimental animal. Over the years, the chinchilla has been used in a wide variety of auditory system experiments and consequently, much is known about its threshold (Miller, 1970; Henderson et al., 1983), psychophysical tuning curves (McGee et al., 1976; Salvi et al., 1982a), thresholds for gap detection (Giraudi et al., 1980) and amplitude modulated noise (Salvi et al., 1982b). These psychophysical results indicate that the chinchilla's hearing capabilities are quite similar to those of humans. The chinchilla is perhaps the most common animal used in noise trauma research even though there is a general consensus that the species is more susceptible to noise trauma than are humans (Trahiotis, 1976). However, phenomenologically the chinchilla is considered to be a suitable model for humans. Thus, the chinchilla was chosen as a reasonable animal model for the blast wave studies presented in this document.

Each animal was anesthetized [IM injection of Telazol® (20 mg/kg)], and made monaural by the surgical destruction of the left cochlea. The monauralization allowed for the testing of hearing function in a single ear. During this surgical procedure, an electrode was permanently implanted near the inferior colliculus for single-ended, near-field recording of the evoked potential (Henderson et al., 1973; Salvi et al., 1982a). The animals were allowed to recover for at least a week before evoked potential testing began.[1]

Two hundred and seventy (270) chinchillas that met the criteria set for acceptable subjects completed the experimental protocol. The following criteria were used to reject an animal from the experimental subject pool: (i) poor preexposure threshold relative to laboratory

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[1] There is no question that destruction of one cochlea alters the efferent interactions between ears that are known to exist in binaural animals. Some recent studies using the quinea pig (Rajan, 1990) have shown that efferent interactions can alter the response of the cochlea to excessive stimulation. However, the results from other laboratories [e.g. Liberman (1990) using the cat] are contradictory. While some differences may be expected on the basis of species differences, species differences alone cannot account for the extent of these disagreements (i.e., Liberman, 1990 vs. Rajan, 1990). The efferent interaction studies mentioned above were performed using relatively low level, short duration, temporary threshold shift producing exposure paradigms. The experiments we are reporting on are completely different (i.e., variable duration, high level, PTS producing exposures where relatively large effects are to be measured). Assuming that the results of Rajan are correct, there is no good evidence that the efferent system will exert the same kind of effects in the type of PTS producing exposure paradigm that we used. If the essentially negative results of Liberman are correct then the whole issue disappears. Another point to note in regard to monauralization is derived from the experiment of Clark and Bohne (1990) in which moderate levels of low frequency noise presented on an intermittent schedule over a long time period were used. Clark and Bohne used some animals which had been surgically monauralized and others in which only the incus was removed to effect a monaural preparation. They found no difference in the results of their noise exposures. Thus, based upon the Clark and Bohne results and the contradictory findings of Liberman and Rajan, there was no compelling reason to alter our experimental protocol regarding surgical monauralization.

norms (see Figure 14); (ii) complications of surgery (e.g., vestibular problems, infection, etc.); (iii) abnormal preexposure tympanograms.

B. Preexposure AEP testing: Hearing thresholds were estimated on each animal using the auditory evoked potential (AEP). The AEP has been shown to be a valid index of hearing threshold in the chinchilla. The correlation between the behavioral and evoked potential measures has been strengthened by directly comparing, in the same animal, estimates of noise-induced behavioral and evoked potential threshold shifts (Henderson et al., 1983; Davis and Ferraro, 1984). There is a close correlation between the behavioral and evoked potential thresholds before, during, and after acoustic overstimulation. In other words, the AEP threshold estimation procedure provides a good estimate of the magnitude of noise-induced hearing loss (see also Hamernik et al., 1990 and Davis et al., 1989). The animals were awake during AEP testing and restrained in a yoke-like apparatus to maintain the animal's head in a constant position within the calibrated sound field (Blakeslee et al., 1978). AEP's were collected to 20 msec tone bursts (5 msec rise/fall time) presented at a rate of 10 per second. A general-purpose computer (Digital Equipment Corporation MicroPDP-11/73) with 12-bit A/D converter (Data Translation 3362), timer (ADAC 1601) and digital interface (ADAC 1632) was used to acquire the evoked potential data and control the frequency, intensity, and timing of the stimulus via a programmable oscillator (Wavetek Model 5100 or Model 23), programmable attenuator (Spectrum Scientific MAT) and electronic switch (Coulbourn Instruments S84-04). The electrical signal from the implanted electrode was amplified (50,000x) and filtered (30 Hz to 3000 Hz) by a Grass P511J biological amplifier and led to the input of the A/D converter where it was sampled at 20 samples per second (50 msec period) over 500 points to obtain a 25 msec sampling window. Each sampled waveform was analyzed for large amplitude artifacts and, if present, the sample was rejected from the average and another sample taken. Averaged AEP's were obtained from 250 presentations of the 20 msec signal. Each waveform was stored on disk for later analysis. A schematic of the main laboratory computer (host) system with which the AEP system interacts and the AEP laboratory is shown in Figures 1 and 2 respectively. Thresholds were measured using an intensity series with 5 dB steps at octave intervals from 0.5 to 16.0 kHz and at the half-octave frequency of 11.2 kHz. Threshold was determined to be one-half step size (2.5 dB) below the lowest intensity that showed a response consistent with the responses seen at higher intensities. The intensity resolution of our method was 5 dB. The average of at least three separate threshold determinations at each frequency obtained on different days was used to obtain the preexposure audiogram.

C. Middle Ear Function: In order to be certain that the blast waves had not altered middle ear function and thus indirectly contributed to the measured threshold changes or to a protective effect for subsequent impulses, tympanometric functions were measured just prior to exposure and immediately following exposure. A Grason-Stadler 1723 Middle Ear Analyser was used to obtain tympanograms at 220 and 660 Hz. The tympanogram will indicate perforations, disarticulations, severe edema, etc. The specific methodology and experimental details can be found in Eames et al. (1975). All data reported here were acquired from animals whose tympanometric functions were normal pre- and postexposure.

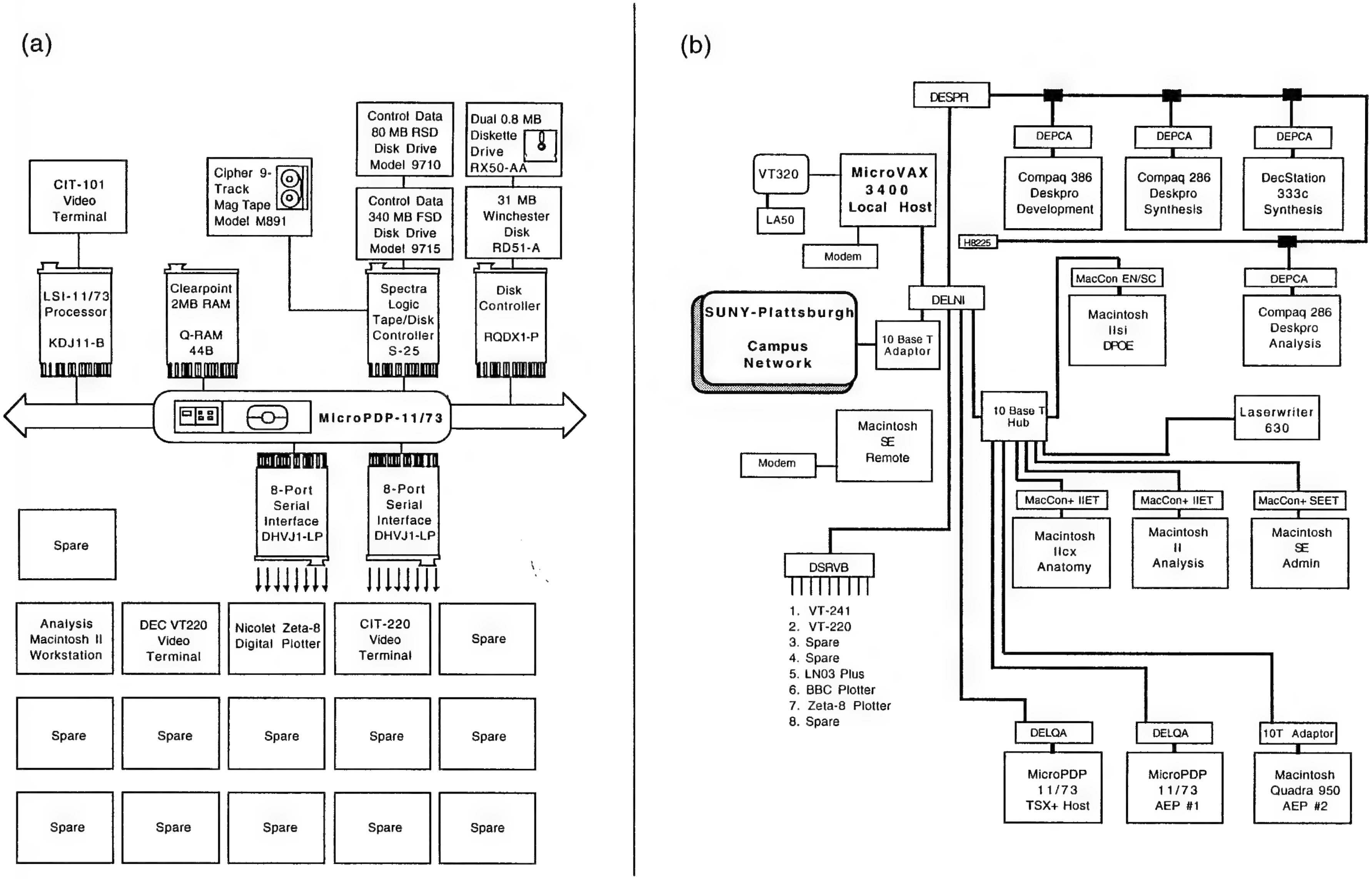

Figure 1. Schematic representations of (a) the Micro-PDP 11/73 time-sharing computer system and (b) the ARL local area network (LAN) with MicroVAX 3400 computer system.

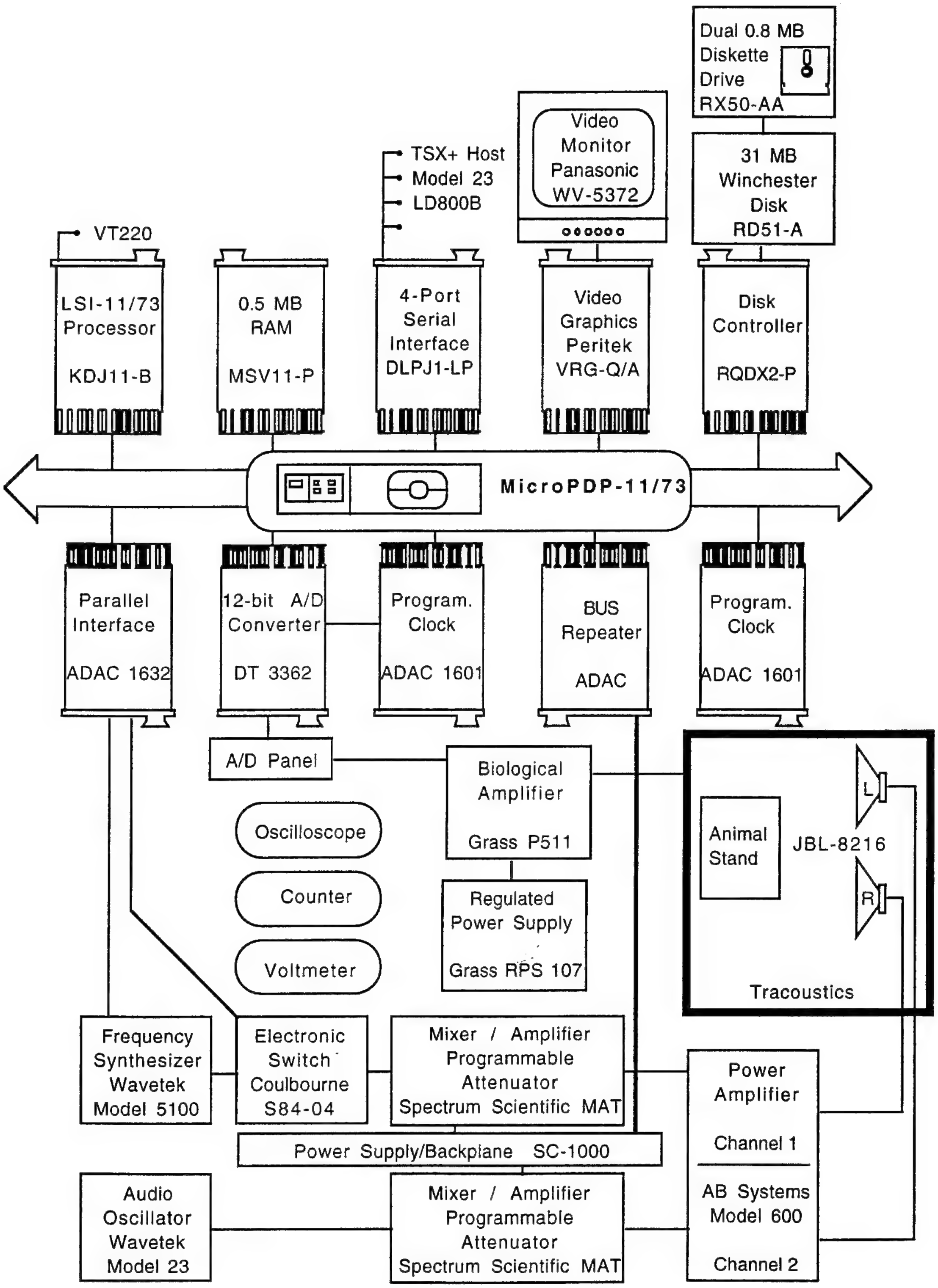

Figure 2. Schematic representation of the auditory evoked potential computer system.

D. Blast Wave Generation, Measurement and Analysis: A conventional bursting-diaphragm shock tube (Source I) and a 3" diameter fast-acting valve-controlled shock tube (Source III) were used to produce the blast wave stimuli. The two sources are described in greater detail in contractor reports ADA 203-854, 206-180, and 221-731.

Basically, these two sources produce blast waves whose peak intensity can be systematically varied from approximately 150 dB through over 180 dB SPL. Source I and Source III were chosen to be used in the generation of the reverberant blast waves because of the differences in the spectra of the two primary (nonreverberant) blast waves produced by these sources [i.e., an octave band A-weighted, spectral peak at 0.25 kHz (Source I) versus 2.0 kHz (Source III)].

Source I is a conventional shock tube which uses a bursting diaphragm to produce a shock wave. A schematic of this facility is shown in Figure 3. The blast waves produced by Source I are a rough approximation of the signature of a 105mm howitzer discharge, while Source III waves are more typical of a smaller bore discharge. The shock tube is coupled to a 6-foot exponential horn having a 4' x 4' exit. Detailed operation and performance characteristics of this source can be found in Hamernik et al. (1973). The Freidlander blast wave produced by this source has an A-duration of approximately 1.5 msec and a spectrum which peaks around 100 Hz.

Source III, the 3-inch Lamont shock tube source, is shown schematically in Figure 4. A cross-sectional view of the "Lamont" driver is shown in Figure 5. The Lamont source uses a relatively simple rapid-acting valve to quickly establish a high pressure discontinuity in the expansion section in order to "drive" the shock front. A force differential generated over the area of the low pressure chamber relative to the high pressure chamber on the rear plate maintains the seal of the high pressure chamber. As the low pressure is gradually reduced, a point is reached where the net force acting on the value reverses direction and the valve rapidly thrusts forward releasing the "slug" of high pressure gas into the expansion section. N_2 was used as the operating gas. The pressure in the high pressure chamber was varied from approximately 100 psig to 1000 psig to achieve peak sound pressure levels (SPL) of the blast wave of from 150 dB to in excess of 160 dB at the exposure location.

The peak SPL of the blast wave from both sources was controlled by systematically adjusting the pressure in the compression section and the distance of the subject from the end of the horn or expansion section. The pressure-time history of the free field blast wave, for calibration purposes, was recorded using a transducer located on the center line of the outlet of the shock tube at the location of the test animal.

In order to produce reverberant conditions, a hard-walled chamber was built. The chamber was built in the approximate shape of a dodecahedron from joined 1/4 inch aluminum plate. The outer shell was constructed in a manner that allowed the rear one-third of the chamber to be opened. If necessary additional reflecting or absorptive surfaces could then be mounted internally to modify the final wave form. This design also allowed easy positioning of a restrained animal in the center of the chamber. The animal was secured to a 4 x 5 inch plate mounted at the end of a 0.5 inch steel post in the center of the 3 foot diameter chamber. The mounting platform allowed the awake and

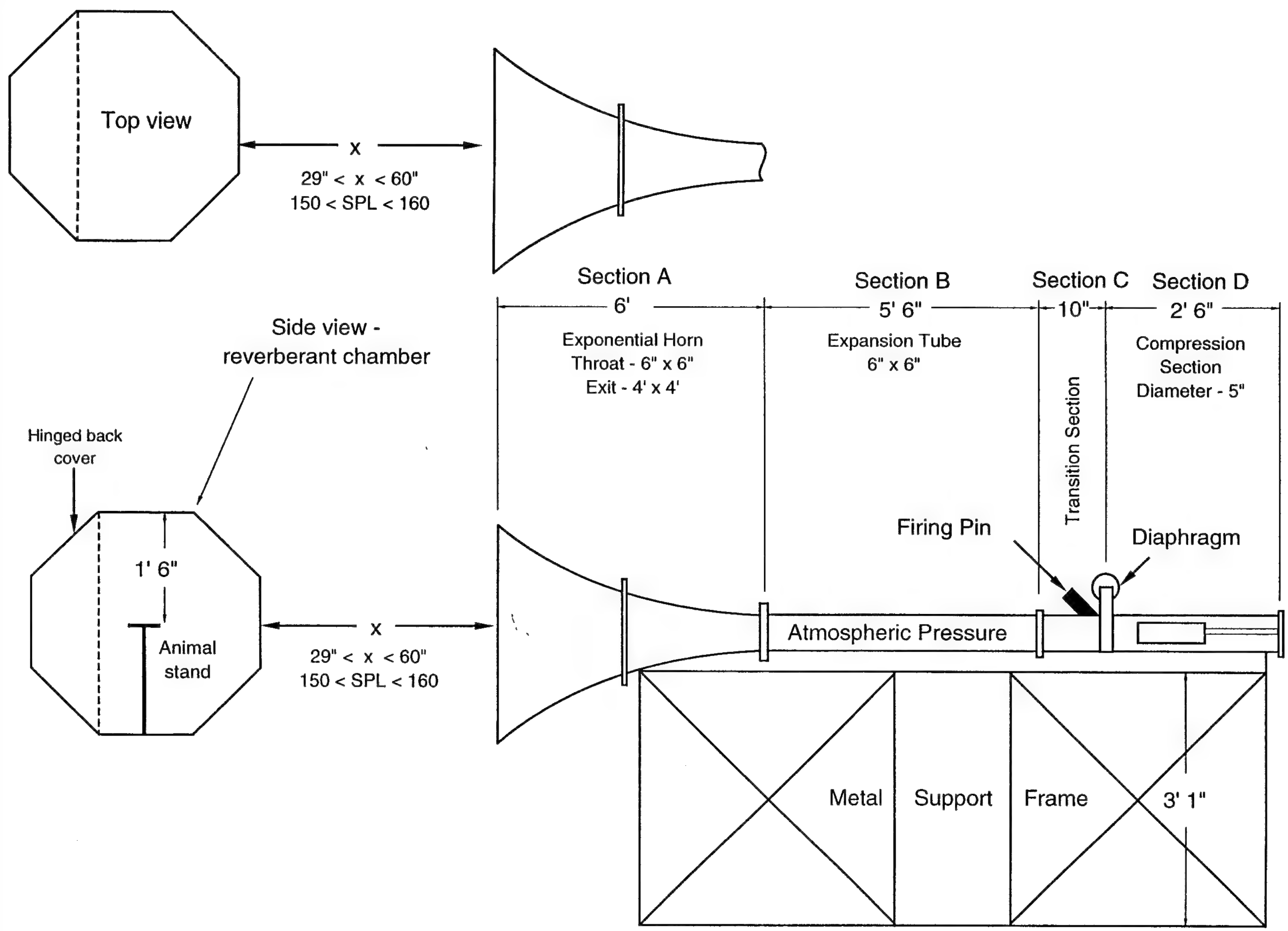

Figure 3. Schematic of the conventional shock tube (Source I) with reverberant chamber.

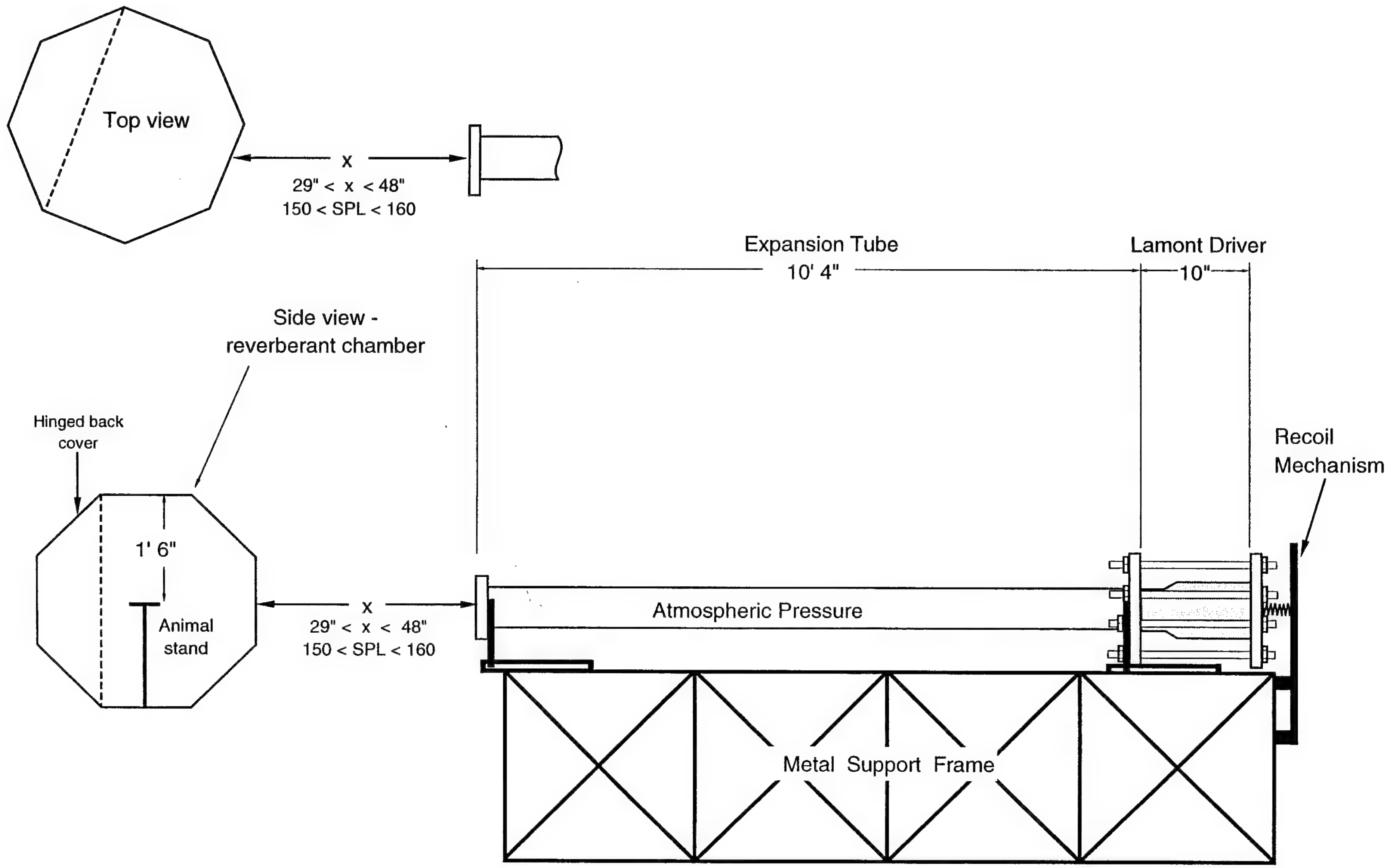

Figure 4. Schematic of "Lamont" shock tube (Source III) with reverberant chamber.

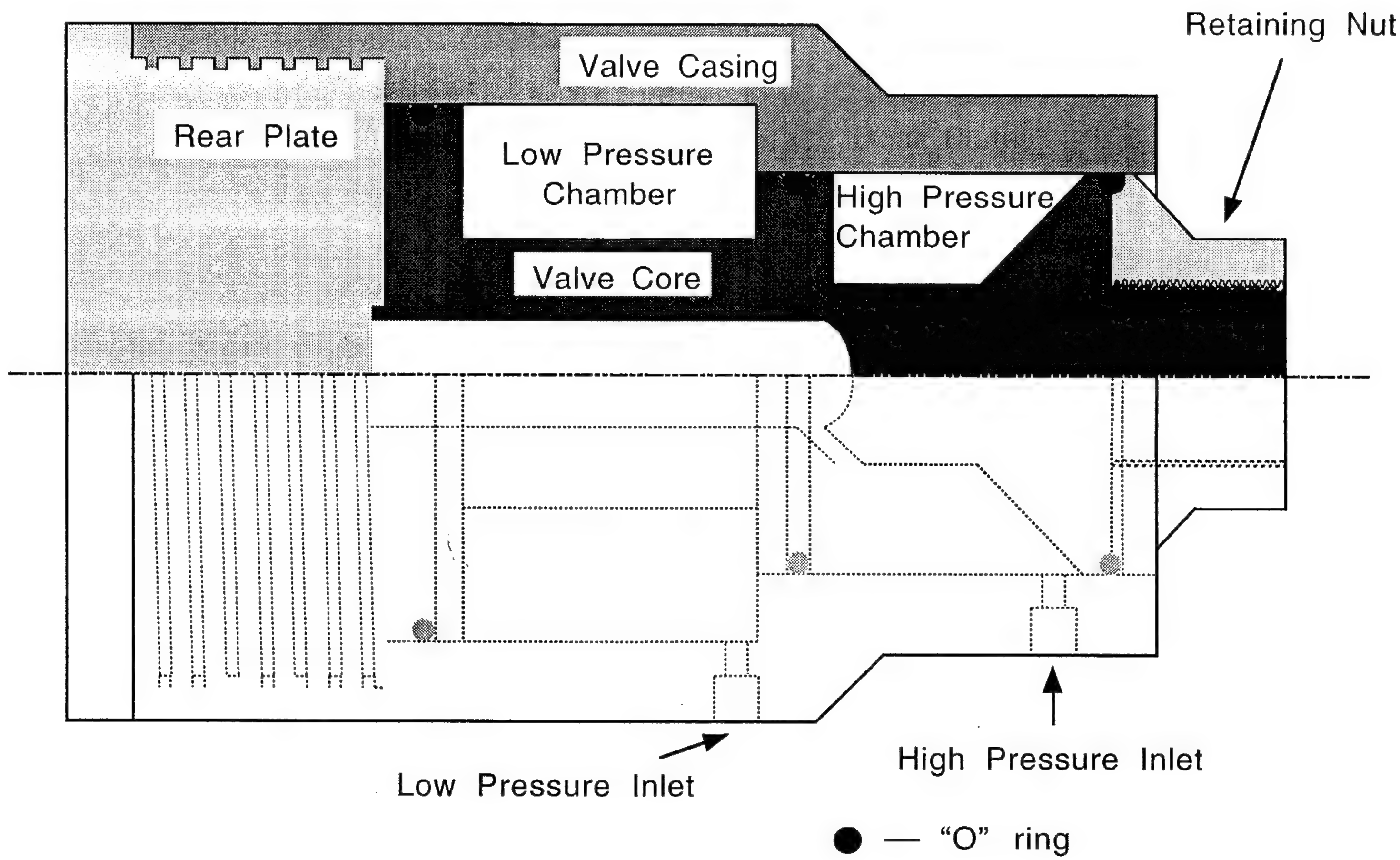

Figure 5. Schematic of the Lamont Fast-acting Valve Used as the Driver in the Source III Shock Tube.

restrained animal to be fixed with its head in the geometric center of the chamber with the experimental ear facing the source (i.e., normal incidence). The chamber was designed with an instrumentation port that allowed calibration to be performed as described in the next section.

E. Calibration of the Chamber: The computer system used in this calibration was a Compaq 286 Deskpro personal computer using the ASYST™ application package (ASYST™ Software Technologies, Inc., Rochester, NY). A schematic of the instrumentation is shown in Figure 6. The blast wave was first digitized and then recorded on mass storage devices (e.g., hard disk or magnetic tape). By using the customized software developed in our laboratory, each digitized blast wave was analyzed to extract information such as the total "acoustic energy" (time integrated pressure squared), energy spectrum, peak and root-mean-square (RMS) sound pressure level (SPL), etc. in accordance with the suggestions of Young (1970). An outline of the procedures used to compute the energy metrics is presented below. Two different types of transducers were used to convert the dynamic acoustic pressure into an electrical signal. The Brüel & Kjær (B&K) 1/8 inch microphone (Type 4138) and the PCB crystal microphone (Model 112A22) were selected because of their ability to record high peak levels and their relatively fast rise times. A B&K microphone preamplifier (Type 2639), a B&K measuring amplifier (Type 2606 or Type 2610), and a PCB six-channel amplifying power unit (Model 483A08) were used to amplify the analog signals from the B&K and PCB microphones respectively. Both transducers yielded identical results. Since the PCB transducers are more rugged and much less expensive, they were used for routine calibration. Performance and calibration of the PCB's, however, was regularly checked with the B&K measurement system. The B&K system was calibrated using a B&K pistonphone. The output signal from the transducer was amplified and, in order to avoid aliasing problems that can occur in analog-to-digital (A/D) conversion, the amplified signals were filtered using an anti-aliasing filter prior to digitizing. The sampling rate of the A/D converter (12-bit) was set at 500 samples per second and the cut off frequency of the anti-aliasing filter was set at 150 kHz (approximately 1/3 of the sampling rate). For each blast wave, 16,384 samples were recorded for later analysis. Software was written using this PC-based system to perform the following tasks: total sound exposure and exposure level calculations (Young, 1970) and spectral analysis using a 4096-point FFT, A-weighted analysis, and P-weighted analysis (Patterson et al., 1993). Thus, for each impact the total as well as weighted sound exposure or exposure level was calculated (i.e., the time integrated, squared sound pressure).

F. Computation of Energy Metrics. The finite-duration pressure-time waveform P_t was uniformly sampled into discrete samples N. Each processed section of the N samples is referred to as a window.

If f_s = sampling rate, and $\Delta t = \frac{1}{f_s}$ then $\Delta f = \frac{f_s}{N}$ is the frequency resolution of the analysis window. The time duration of the window is:

$$t_w = N \cdot \Delta t = \frac{N}{f_s}$$

Let P_t represent the temporal values of the N samples of the pressure-time waveform, and let P_f be the fast Fourier transform (FFT) values of P_t.

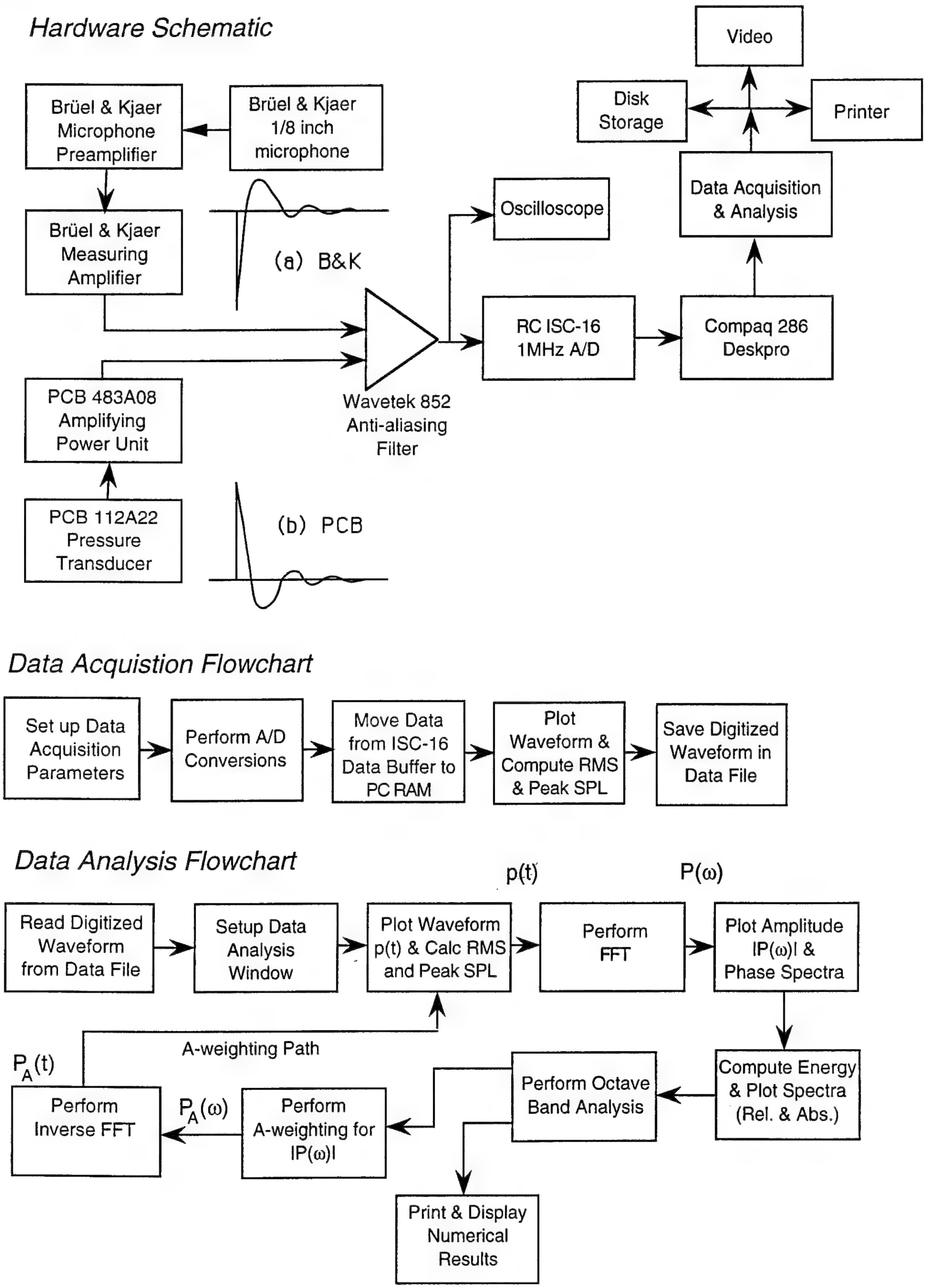

Figure 6. Configuration of the MS-DOS PC-Based Data Acquisition and Analysis System.

The FFT of P_t is then $$P_f = \sum_{n=0}^{N-1} P_t \; e^{-j2\pi kn/N} \qquad k = 0,1,2,\ldots,N-1$$

and the inverse FFT is $$P_t = \frac{1}{N} \sum_{k=0}^{N-1} P_f \; e^{j2\pi kn/N} \qquad n = 0,1,2,\ldots,N-1.$$

An energy metric (i.e., Pressure^2 x time) can be obtained from both time and frequency domains through the application of Parseval's theorem. That is,

$$\sum_{n=0}^{N-1} P_t^{\,2} = \sum_{n=0}^{N-1} P_t \, P_t^{*} \qquad \text{(* indicates complex conjugate)}$$

$$= \sum_{k=0}^{N-1} \frac{1}{N^2} \sum_{k_1=0}^{N-1} P_f \; e^{j2\pi k_1 n/N} \sum_{k_2=0}^{N-1} P_f{}^{*} \; e^{-j2\pi k_2 n/N}$$

$$= \frac{1}{N^2} \sum_{k_1=0}^{N-1} \sum_{k_2=0}^{N-1} P_f \, P_f{}^{*} \sum_{k=0}^{N-1} e^{-j2\pi(k_2-k_1)n/N}$$

$$= \frac{1}{N^2} \sum_{k_1=0}^{N-1} \sum_{k_2=0}^{N-1} P_f \, P_f^{*} \, N \cdot \delta(k_1-k_2) = \frac{1}{N} \sum_{k=0}^{N-1} |P_f|^2$$

$$\left(\text{since} \sum_{n=0}^{N-1} e^{j2\pi(k_1-k_2)n/N} = \begin{cases} 0 & k_1 \neq k_2 \\ N & k_1 = k_2 \end{cases} \right).$$

The total sound energy ($Pa^2 \cdot sec$) is equal to the sum of the squared pressure amplitudes over the time interval. The total energy also can be obtained as the sum of the squared spectrum amplitude over all frequencies from Parseval's theorem. That is,

$$\sum_n P_t^{\,2} \cdot \Delta t = \frac{1}{N} \sum_k |P_f|^2 \, \Delta t = \frac{1}{N^2} \sum_k |P_f|^2 \, \Delta t \cdot N$$

$$= \frac{1}{N^2} \sum_k |P_f|^2 \cdot N/f_s = \frac{1}{N^2} \sum_k |P_f|^2 \,/\, \Delta f$$

The total sound exposure level (SEL) in dB (re: t_0 = 1 sec, P_0 = 20 µPa) is:

$$\text{SEL} = 10 \log \frac{\sum_{n} P_t^{\,2} \Delta t}{P_0^{\,2} t_0} \qquad \text{in the time domain, while}$$

$$\text{SEL} = 10 \log \frac{\frac{1}{N^2} \sum_{k} |P_f|^2 / \Delta f}{P_0^{\,2} t_0} \qquad \text{in the frequency domain.}$$

The pressure square spectrum level is:

$$10 \log \frac{\frac{1}{N^2} \; |P_f|^2}{P_0^{\,2}} \qquad (P_0 = 20\ \mu\text{Pa})$$

The octave band sound exposure ($Pa^2 \cdot$ sec) can be obtained from the sum of the squared spectrum amplitude over the octave band range. That is,

$$\frac{1}{N^2} \sum_{\text{octbands}} |P_f|^2 \;/\; \Delta f$$

The octave band sound exposure level in dB (re: $t_0 = 1$ sec, $P_0 = 20$ μPa) is:

$$10 \log \frac{\frac{1}{N^2} \sum_{\text{octbands}} |P_f|^2 \;/\; \Delta f}{P_0^{\,2} t_0}$$

The A-weighted and P-weighted exposure energies (i.e., $Pa^2 \cdot$ sec) were obtained as follows:

$|P_{fA}| = |P_f| \; |W_{fA}|$ Where W_{fA} is the A-weighting function

$|P_{fP}| = |P_f| \; |W_{fP}|$ and W_{fP} is the P-weighting function (Patterson et al., 1993).

The total weighted energy ($Pa^2 \cdot$ sec) is, therefore,

$$\frac{1}{N^2} \sum_{k} |P_{fA}|^2 \;/\; \Delta f \qquad \text{(A-weighting)}$$

$$\frac{1}{N^2} \sum_{k} |P_{fP}|^2 \;/\; \Delta f \qquad \text{(P-weighting)}$$

The total sound exposure level in dB (re: $t_0 = 1$ sec, $P_0 = 20$ μPa) is:

$$\text{SEL}_A = 10 \log \frac{\frac{1}{N^2} \sum_{k} |P_{fA}|^2 / \Delta f}{P_0^{\,2} t_0} \qquad \text{(A-weighting)}$$

$$SEL_P = 10 \log \frac{\frac{1}{N^2}\sum_k |P_{fP}|^2/\Delta f}{P_0^2 t_0} \quad \text{(P-weighting)}$$

The weighted pressure square spectrum level in dB (re: $P_0 = 20\ \mu Pa$) is given by:

$$10 \log \frac{\frac{1}{N^2}|P_{fA}|^2}{P_0^2} \quad \text{(A-weighting)}$$

$$10 \log \frac{\frac{1}{N^2}|P_{fP}|^2}{P_0^2} \quad \text{(P-weighting)}$$

G. Exposure of Animals: For a given exposure condition, each chinchilla was exposed at the same calibrated location of the reverberant chamber. During exposure the animal was unanesthetized but immobilized in a leather harness (Patterson et al., 1986). The right pinna was folded back and fixed in place to insure that the entrance of the external meatus is not obstructed and the position of the entire animal was adjusted so that the cross sectional plane of the meatus was oriented parallel to the plane of the advancing shock front (i.e., a normal incidence). Each experimental group consisted of fifteen (15) animals. Each animal was individually exposed to one of the following exposure conditions: Source I or Source III; 150, 155, or 160 dB peak SPL; 1, 10, or 100 impulses presented at the rate of 1/min. This combination of two sources, three intensities, and three numbers of impulses yielded a total of 270 animals distributed in 18 groups. The interstimulus internal (ISI) was fixed at the rate of 1/min for two reasons: (1) our earlier results indicated that exposure paradigms with ISI's in the range 10/min through 1/10 min do not in general produce systematic statistically significant different results (Hamernik et al., contractor report ADA 241-117, 1991); and (2) to attempt to minimize the number of animals and time expenditure by focusing first on the most important energy determining variables of peak level and number. An ISI of 1/min also represents a reasonable approximation to many situations encountered in the field.

H. Postexposure AEP Testing: After the exposure was completed, AEP threshold recovery functions were measured once at 0.5, 2.0 and 8.0 kHz at 0, 2, 8, 24 and 240 hours after removal from the noise (using the same method as described for preexposure testing). After at least 30 days postexposure, final audiograms were constructed using the average of three separate AEP threshold determinations at each of the seven preexposure frequencies. Permanent threshold shift (PTS) was defined as the difference between the postexposure and preexposure thresholds at each individual test frequency.

I. Cubic Distortion Product Otoacoustic Emmissions (3DPE): The Virtual Model 330 Otoacoustic Emissions Test Instrument was used to collect 3DPE data on a subset of the animals in the blast wave exposure protocol. Data collection consisted of two parts:

(i) The establishment of a normative data base consisting of 102 preexposure (monaural) animals from which 3DPE data were acquired as a

function of frequency at two intensities and 104 animals from which 3DPE input/output functions were acquired at six frequencies.

(ii) The collection of DPEgrams (i.e., the 3DPEs as a function of frequency) and I/O functions (i.e., 3DPEs as a function of primary level at a given frequency $f = \sqrt{f_1 f_2}$) from 47 animals exposed to various blast waves.[2] In addition, a subset (N = 9) of the animals in (i) above were tested in the right ear with the Model 330 prior to surgery (i.e. while still binaural) and compared to the mean data obtained from the monaural animals. These 9 binaural animals were also tested over a long period of time (25 days) to examine test-retest reliability. (Note: Despite the restraining device, some animals would either continually struggle or vocalize while restrained. Reliable 3DPE data could not be easily obtained from such animals. Thus, animals that exhibited such behavior were not tested thus further limiting the subject pool from which otoacoustic emissions data could be obtained.)

3DPE Experimental protocol: During all 3DPE testing, the animals were awake and restrained. The 3DPE collection parameters were: eight time averages; $f_2/f_1 = 1.22$ where f_1 and f_2 are the lower and upper primary tone frequencies respectively; DPEgrams measured using 55 and 65 dB SPL primary tones with $L_1 = L_2$ (L = primary tone level); 3DPE plotted as a function of $\sqrt{f_1 f_2}$, where $500 \leq \sqrt{f_1 f_2} \leq 8000$ Hz, at 1/6-octave steps; I/O functions obtained in 5 dB steps at $\sqrt{f_1 f_2} = 1.0$, 2.0, 3.0, 4.0, 6.0, and 8.0 kHz.

Each DPEgram and each I/O function was measured five separate times (except for the subset that underwent the test-retest reliability study), before exposure and 30 days after exposure. The mean was accepted as that subjects pre- or postexposure DPEgram or I/O function.

3DPE Test-Retest Protocol: A group of nine (9) binaural animals underwent the following test protocol:

DPEgram; using 55 dB primaries, two DPEgrams were collected, one after the other. The probe was removed and reinserted and a third DPEgram was collected. The above protocol was repeated for 65 dB primaries. The probe was removed and reinserted and a single set of I/O functions was then collected. The procedure was repeated on 25 different days. Each animal then underwent monauralization surgery and following a two-week recovery period the same DPEgram and I/O data collection protocol was repeated on five consecutive days and the data was entered into the normative data pool of monaural animals. [Note: During the postsurgery recovery period, two of the original nine animals developed problems and were eliminated from the postsurgery data collection paradigm (i.e., the postsurgery subject pool consisted of an N = 7.) This protocol allowed for various analysis of normal 3DPE functions as presented in the results section.

3DPE Following Blast Wave Exposure: Forty-seven (47) animals, some of which were a subset of the normative study, were exposed to various

[2] The acquisition of 3DPE data was not part of the original statement of work for this contract. When equipment became available in the last half of the contract tenure, the decision was made to obtain 3DPE data on as many of the remaining animals as possible. Thus, sample sizes and exposure groups are not always consistent with the original experimental design.

blast waves. [Note: Preexposure 3DPEs were not collected on some of these animals since the emissions laboratory was not set up. Thus pre/post comparisons of the exposed animals are made with the normative data base.] Thirty days following exposure three DPEgrams using 65 and 55 dB primaries with $L_1 = L_2$ and three sets of I/O functions were recorded on three different days. The mean was used as the 30-day postexposure DPEgram or I/O function for comparison with normative preexposure data.

J. Cochlear Histology: Following postexposure audiometric and emissions testing, the animals were euthanatized under anesthesia by decapitation and the cochleas immediately removed and fixed. The cochleas were dissected and the status of the sensory cell population evaluated using conventional surface preparation histology (Engstrom et al., 1966). Briefly, the stapes was removed and the round window membrane opened to allow transcochlear perfusion, via the scala tympani/scala vestibuli, with cold 2.5% glutaraldehyde in veronal acetate buffer at 7.3 pH (605 mOsm). Postfixation was performed on the following day with one percent osmium tetroxide in veronal acetate buffer (pH 7.3) for 30 minutes. The cochleas were then dissected and the entire sensory epithelium was mounted in glycerin on glass slides. The status of sensory and supporting cells were evaluated with Nomarski differential interference contrast microscopy and entered into a data base on a laboratory computer (Digital Equipment Corporation MicroPDP-11/73 and Macintosh IIcx). Standard cochleograms were then constructed by computing the percent sensory cell loss across the length of the cochlea in 0.24 mm steps. These cell loss figures were then converted into percent loss over octave bands centered at the audiometric test frequencies along the length of the cochlea and correlated with the frequency-place map of the chinchilla cochlea constructed by Eldredge et al. (1981). A schematic of the morphometric system is shown in Figure 7.

Quantitative histology of the cochlea, relating sensory cell populations to frequency specific locations on the basilar membrane, is considered a necessary addition to audiometric measures when developing exposure standards. Histology provides an alternate measure of pathology which should correlate with functional measures. However, threshold measures represent only a single dimension of hearing. While traditionally considered to be the most basic measure, thresholds may not always reflect the extent of pathology (See, for example, Eldredge et al., 1973 or Hamernik et al., 1989).

K. Descriptive and Inferential Statistics and Data Archive: The descriptive analysis of the data from these experiments consisted of (a) a complete description of the raw data and group means, standard deviations, and standard errors; (b) graphical representation of all audiometric and otoacoustic emissions data; (c) tabular and graphical representation of individual histological summaries; and (d) group summaries of the histological analyses. These descriptive measures are included in two appendices submitted to the contract officer's representative (COR) along with the interim and final reports.

Descriptive measures reported in the final report include (a) a tabulation of unweighted and weighted energy metrics describing the acoustic analysis of the noise exposure stimuli; (b) figures illustrating the time history and spectra of the noise exposure stimuli; and (c) figures describing the average value of dependent variables

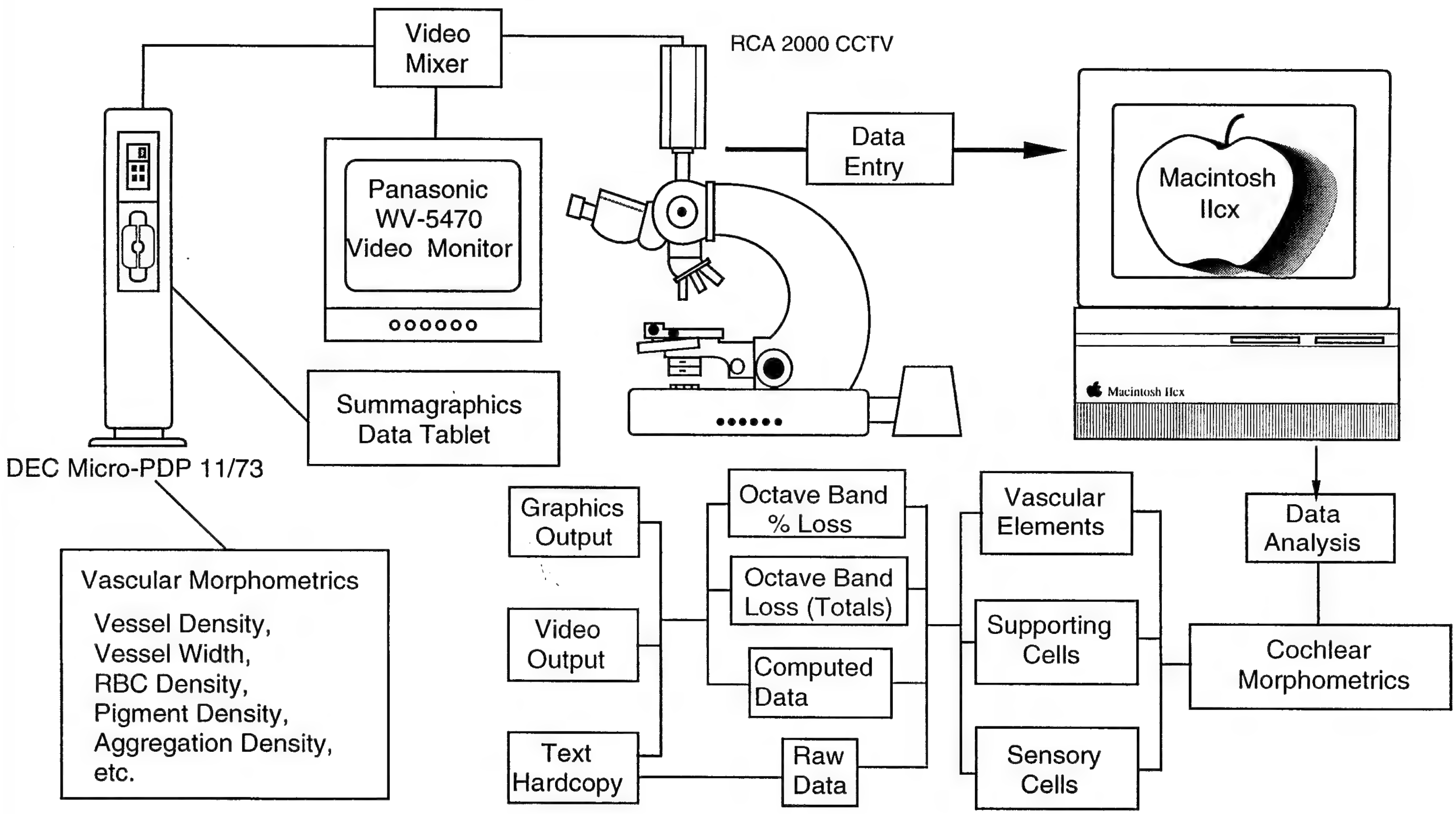

Figure 7. Anatomy Laboratory Temporal Bone Morphometric Analysis Systems.

grouped in logical orders to allow visual comparisons of the effects of the independent variables (e.g., peak pressure, noise source, number of impulses) on the dependent variables (TS_{max}, PTS, percent sensory cell loss, DPEgrams, and 3DPE I/O functions).

Because of the limited sample sizes and the preliminary nature of the DPEgram and 3DPE I/O functions, inferential statistics were not applied to the otoacoustic emission data.

The inferential statistics presented in the interim report included three-way mixed-model analyses of variance with repeated measures on one factor (frequency). In these analyses, the between groups factors of peak pressure and number of impulses allowed the description of statistically significant effects and interactions within the three-way ANOVA. The complete experimental design for the final report includes two sources (Source I and Source III); three peak pressures (150, 155, and 160 dB peak SPL); three numbers of impulses (1, 10, and 100); and three, seven, or eight frequencies as a within groups factor (depending on the dependent variable being analyzed). A set of parallel analyses to that performed for the interim report would involve four-way mixed-model analyses of variance with repeated measures on one factor. The summary tables for these analyses are presented in Table 11.

However, when considering the summary tables from these analyses (four main effects, six two-way interactions, four three-way interactions, and one four-way interaction) it is apparent that the interpretation of these analyses can rapidly become unwieldy. This is especially true when one has to consider that the presence of an interaction effect means that the main effects may not be directly interpretable. For example, the four-way interaction of noise source, peak pressure, number of impulses, and test frequency is statistically significant in the analysis of permanent threshold shifts ($F = 1.66$, $df = 24/1482$, $p < .05$). This interaction means that there are statistically significant differences between the groups, but that these differences depend on what noise source, peak pressure, number of impulses to which the subject was exposed as well as at which test frequency the PTS was measured.

One can observe that a comparison of the figures describing the dependent variable provides much more insight than does the analysis of variance summary table. For example, simply looking at Figures 22 and 23 (below) provides a better understanding the effects of the independent variables than studying the appropriate table in Table 11. An observation of the figures suggest that (1) there is little PTS resulting from only one blast wave; (2) Source III causes more PTS than Source I; (3) higher peak SPL impulses cause more PTS than lower peak SPL impulses; (4) 100 blast waves cause more hearing loss than 10 or 1 blast wave(s); (5) while losses, when they appear, seem to be at all or most test frequencies, some frequencies are effected more than others; and (6) each of these observations are conditional upon the level of the factor of the other dependent variables (i.e., interactions exist). Therefore, because of the complexity of the mixed-model analyses of variance coupled with the large treatment effects (e.g., > 40 dB differences in PTS among groups), the statistical analyses of most of the dependent variables will not be discussed in the body of the report. Rather, interested readers may refer to the analysis of variance summary tables attached to the report (Table 11).

IV. RESULTS AND DISCUSSION

A. Documentation of the Noise Stimulus. As described above, the blast wave stimuli were produced by two different sources, Source I and Source III. Each of these sources, for the same peak SPL produced a waveform having a different temporal and, hence, spectral structure. Following the suggestions of Young (1970), the time-integrated squared sound pressure (or level re: 20 µPa) and its equivalent Fourier spectral representation was computed and is shown graphically in Figures 8 through 13. The waveforms shown in these figures from which spectra and energy metrics were computed were aquired from recordings taken just in front of the entrance of the external meatus, with the animal mounted in the exposure position. Numeral values of the energy metrics, either total or octave band values are shown in Tables 1 through 4 for each of the six waveforms. The pressure-time histories of each of the six impulses are shown as insets in Figures 8 and 11. A- and P-weighting functions (Patterson et al., 1993) were applied to each spectrum. The graphical and tabular results are presented in Figures 9, 10, 12 and 13, and in Tables 3 and 4 respectively, in terms of relative and absolute levels.

B. Preexposure Evoked Response Hearing Thresholds. Preexposure thresholds for the 18 groups (total N = 270) of chinchillas exposed to reverberant blast waves is shown in Figure 14. The preexposure thresholds were analyzed for differences using two-way mixed model analyses of variance with repeated measures on one factor (frequency). The analyses were performed using SPSS release 4 on a Digital Equipment Corporation VAX 6000 series computer system running VMS version 5.5-2. Unless otherwise noted, the probability of a Type I error was set at 0.05. The analysis of mean preexposure thresholds from groups exposed to reverberant impulses from Source I revealed a statistically significant main effect of frequency ($F = 518.01$, $df = 6/750$, $p < .05$) which was expected based upon our knowledge of the chinchilla audiogram (Fay, 1988). The main effect of experimental group was statistically significant ($F = 2.50$, $df = 8/125$, $p < .05$) as was the interaction of group and frequency ($F = 2.87$, $df = 48/750$, $p < .05$). The largest differences between groups amounted to 10 to 15 dB which is well within the range of threshold values presented in a number of different reports summarized by Fay (1988). Since threshold shift data are used as the measure of audiometric results in the present study (as opposed to shifted thresholds), the small differences in mean preexposure thresholds should not have any appreciable effects on the subsequent analyses.

The analysis of mean preexposure thresholds from groups exposed to reverberant impulses from Source III revealed a statistically significant main effect of frequency ($F = 325.21$, $df = 6/750$, $p < .05$). The main effect of experimental group was not statistically significant ($F = 0.88$, $df = 8/125$) nor was the interaction of group and frequency ($F = 1.36$, $df = 48/750$). Summaries of the statistical results of the preexposure AEP thresholds are presented in Tables 5 and 6.

C. Preexposure 3DPE Functions. The mean DPEgram based upon 102 monaural (left cochlea destroyed) chinchillas and the mean I/O functions based upon 104 monaural chinchillas are shown in Figures 15 and 16. 3DPE data at frequencies $f = \sqrt{f_1 f_2} \leq 1.0$ kHz are in general not distinguishable from the noise floor. Above 1.0 kHz, the 3DPE is robust

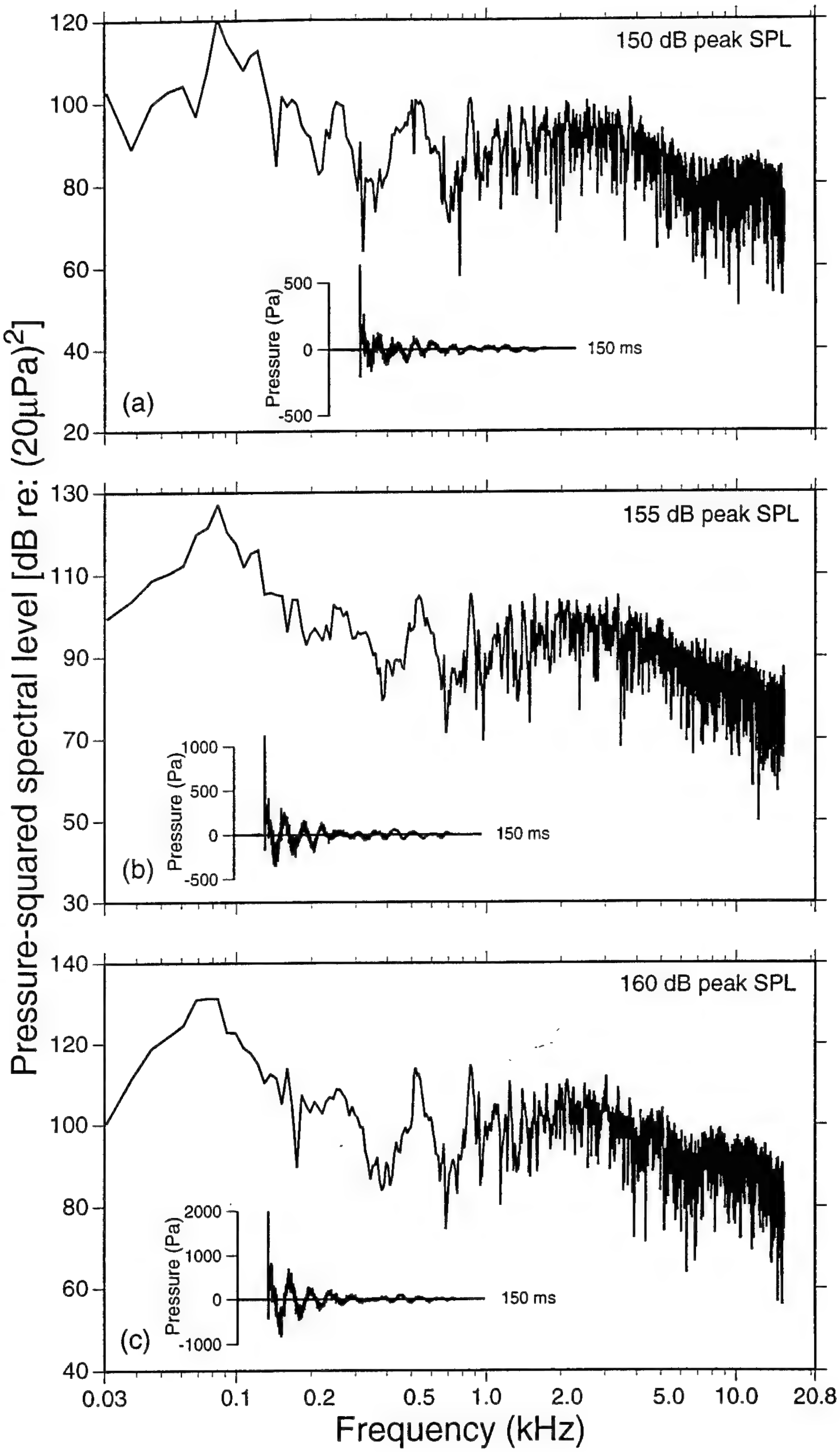

Figure 8. The unweighted frequency spectra and pressure-time waveforms for the (a) 150 dB, (b) 155 dB and (c) 160 dB peak SPL reverberant blast waves produced by Source I. (Stimulus measured with the animal in place.).

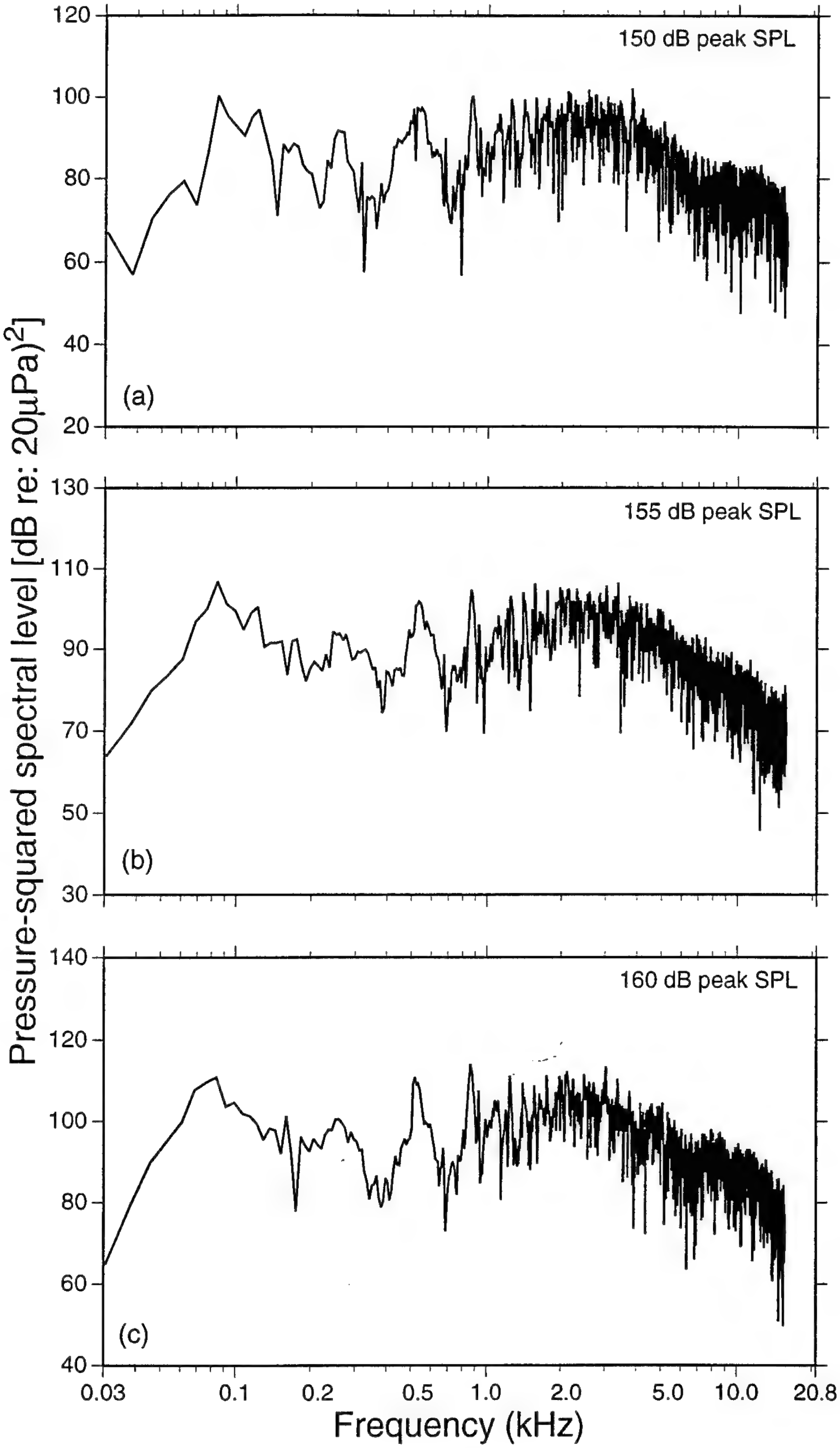

Figure 9. The A-weighted frequency spectra for the (a) 150 dB, (b) 155 dB and (c) 160 dB peak SPL reverberant blast waves produced by Source I.

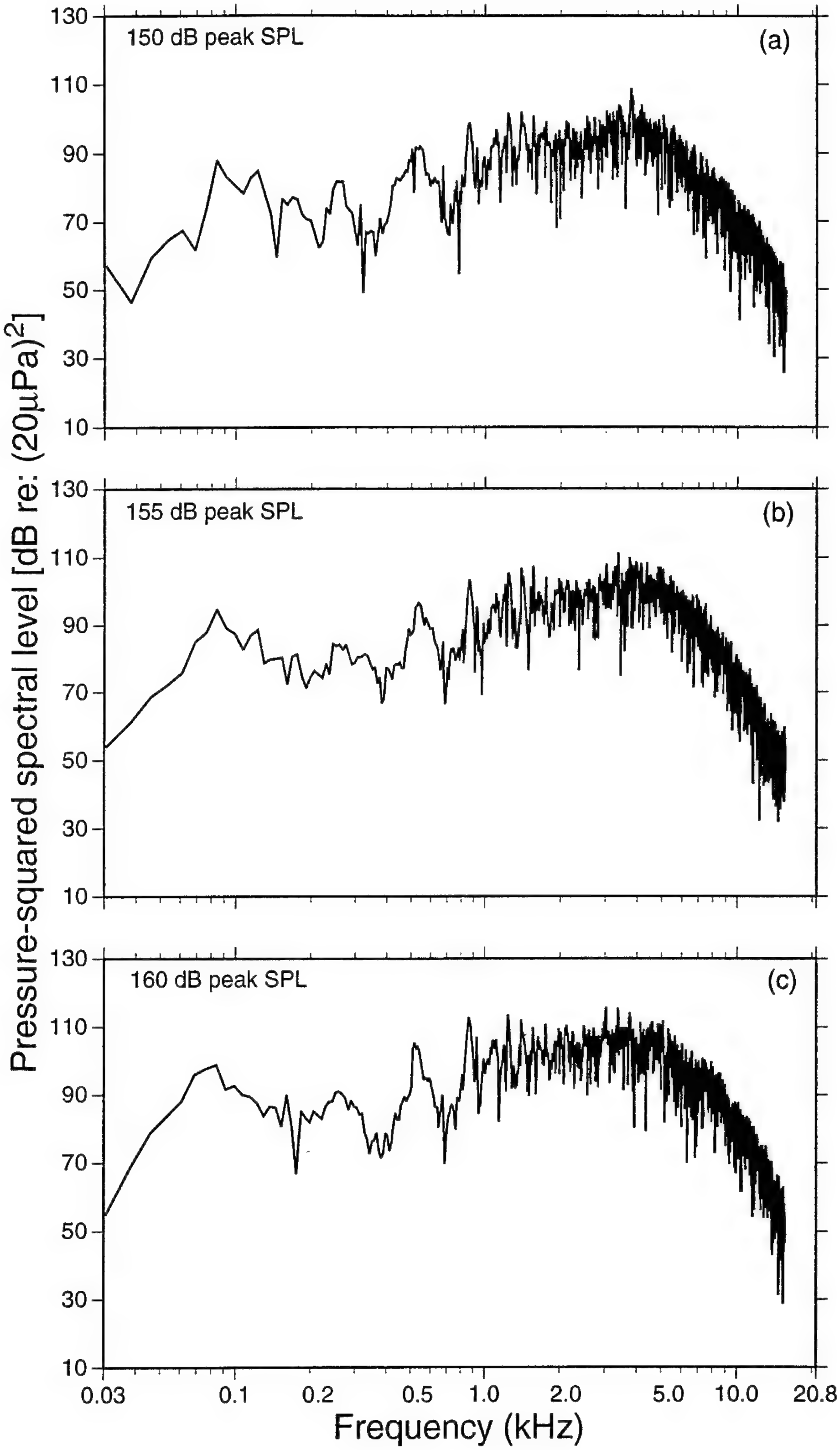

Figure 10. The P-weighted frequency spectra for the (a) 150 dB, (b) 155 dB and (c) 160 dB peak SPL reverberant blast waves produced by Source I.

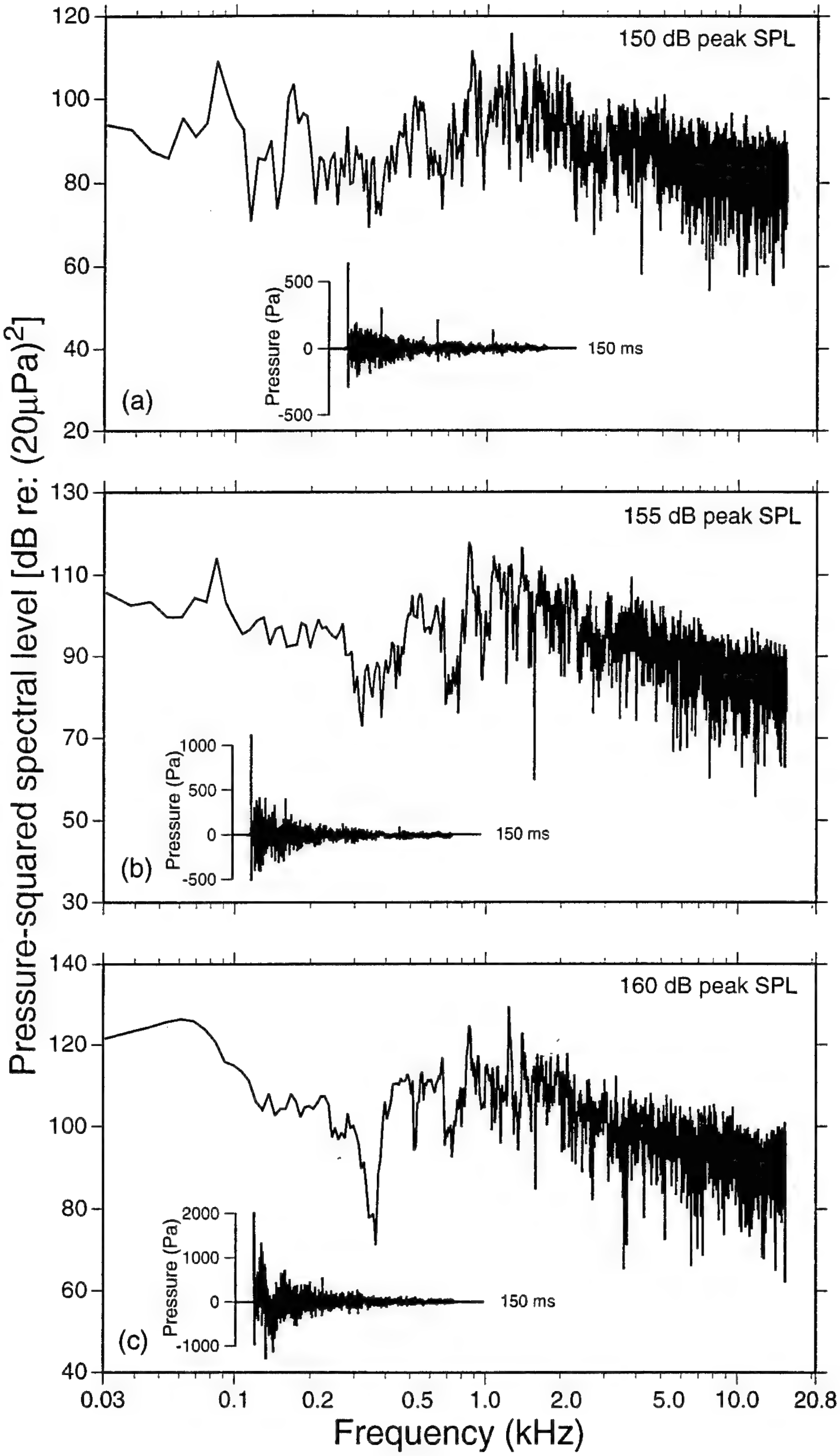

Figure 11. The unweighted frequency spectra and pressure-time waveforms for the (a) 150 dB, (b) 155 dB and (c) 160 dB peak SPL reverberant blast waves produced by Source III. (Stimulus measured with the animal in place.).

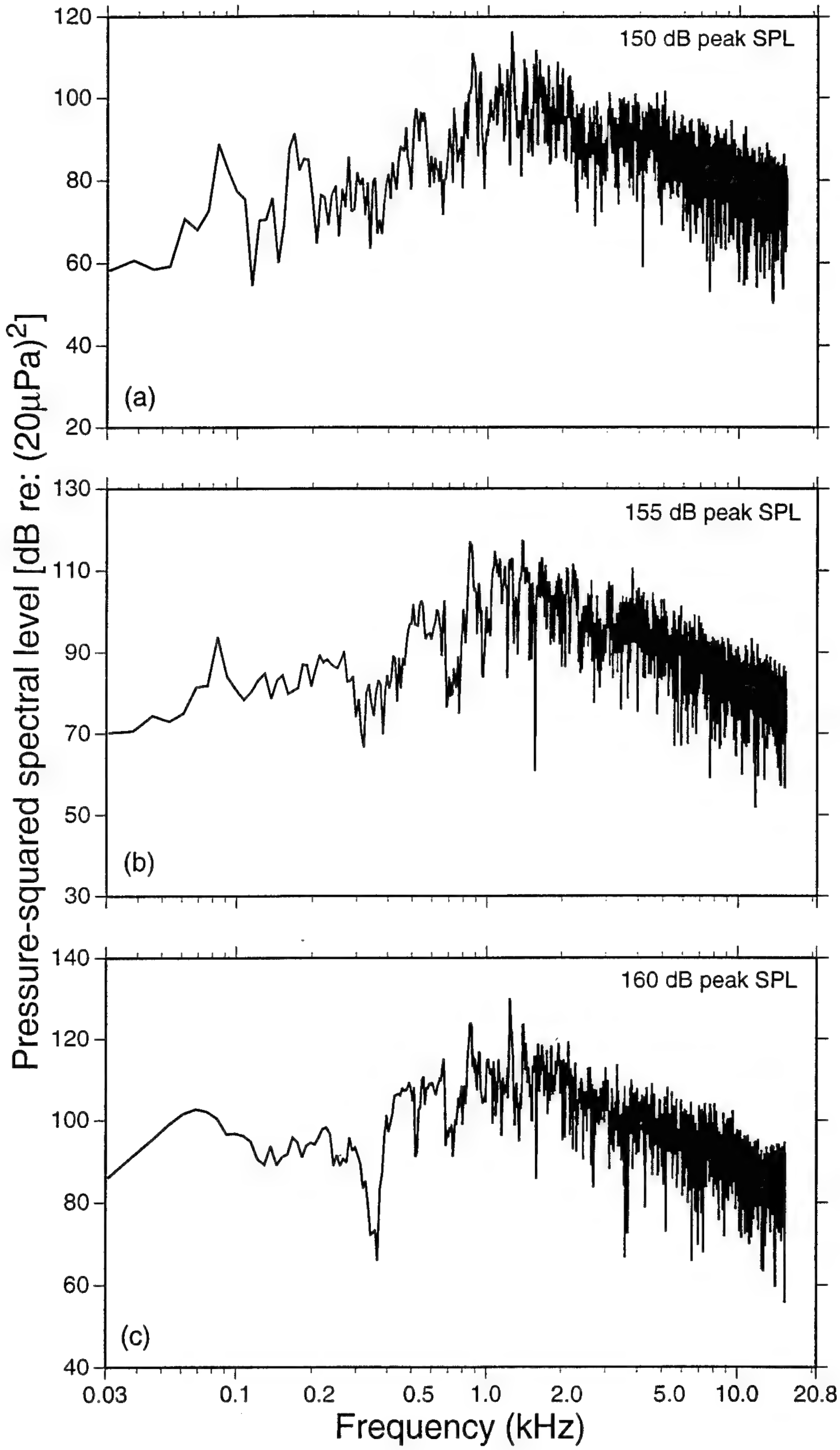

Figure 12. The A-weighted frequency spectra for the (a) 150 dB, (b) 155 dB and (c) 160 dB peak SPL reverberant blast waves produced by Source III.

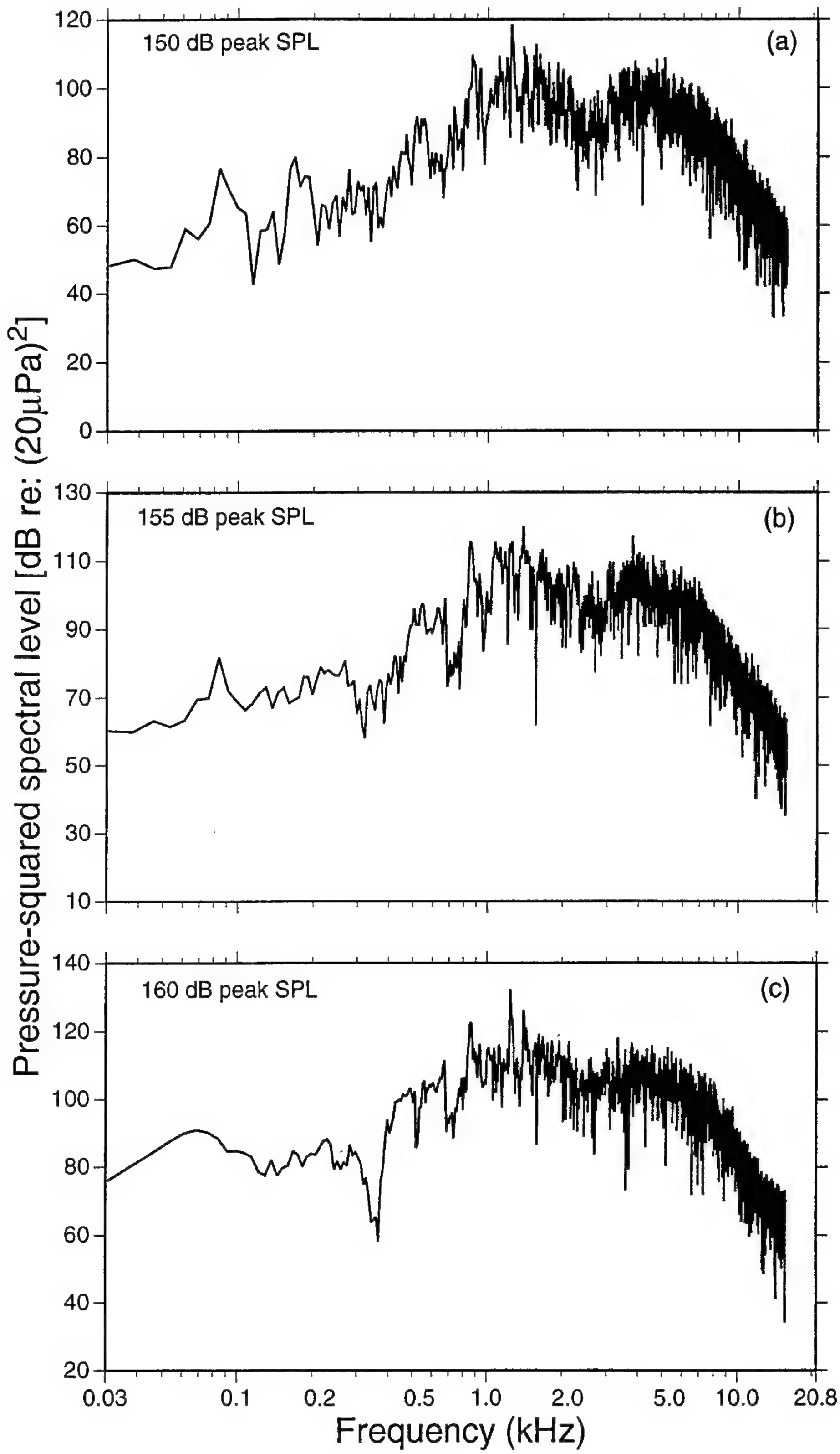

Figure 13. The P-weighted frequency spectra for (a) 150 dB, (b) 155 dB and (c) 160 dB peak SPL reverberant blast waves produced by Source III.

Table 1. The total unweighted and weighted "energy" $\int P^2(t)\,dt$ ($Pa^2 \cdot sec$) for the reverberant blast waves produced by each of the two sources at the three peak levels and the three exposure conditions (1, 10, or 100 blast waves).

Source I	Unweighted	A-weighted	P-weighted
150 dB peak SPL			
1X	196.1	79.7	164.8
10X	1960.5	796.9	1647.6
100X	19605.2	7968.8	16475.7
155 dB peak SPL			
1X	774.7	225.3	472.3
10X	7746.5	2252.7	4723.1
100X	77465.4	22527.3	47230.6
160 dB peak SPL			
1X	3452.3	849.1	1431.1
10X	34523.1	8491.1	14310.8
100X	345230.7	84911.0	143107.9

Source III	Unweighted	A-weighted	P-weighted
150 dB peak SPL			
1X	227.0	238.9	419.1
10X	2269.6	2389.2	4191.4
100X	22696.5	23892.0	41913.6
155 dB peak SPL			
1X	753.7	797.2	1324.3
10X	7537.3	7971.5	13243.3
100X	75373.4	79715.4	132433.3
160 dB peak SPL			
1X	5034.1	4016.2	5825.0
10X	50341.4	40162.2	58249.7
100X	503413.8	401621.7	582496.8

Table 2. The total sound exposure levels (dB SEL) for the reverberant blast waves produced by the two sources at three peak levels and the three exposure conditions (1, 10, or 100 blast waves).

	Source I		
	Unweighted	A-weighted	P-weighted
150 dB peak SPL			
1X	116.9	113.0	116.1
10X	126.9	123.0	126.1
100X	136.9	133.0	136.1
155 dB peak SPL			
1X	122.9	117.5	120.7
10X	132.9	127.5	130.7
100X	142.9	137.5	140.7
160 dB peak SPL			
1X	129.4	123.3	125.5
10X	139.4	133.3	135.5
100X	149.4	143.3	145.5

	Source III		
	Unweighted	A-weighted	P-weighted
150 dB peak SPL			
1X	117.5	117.8	120.2
10X	127.5	127.8	130.2
100X	137.5	137.8	140.2
155 dB peak SPL			
1X	122.8	123.0	125.2
10X	132.8	133.0	135.2
100X	142.8	143.0	145.2
160 dB peak SPL			
1X	131.0	130.0	131.6
10X	141.0	140.0	141.6
100X	151.0	150.0	151.6

Table 3. The unweighted and weighted octave band $\int P^2(t)\,dt$ ($Pa^2 \cdot sec$) for a single reverberant blast wave produced by each of the two sources at the three peak levels.

	Source I			Source III		
150 dB peak SPL	Unwtg.	A-Wtg.	P-Wtg.	Unwtg.	A-Wtg.	P-Wtg.
< 0.125 kHz	79.27	0.59	0.04	5.54	0.04	0.00
0.125 kHz	46.28	0.98	0.07	3.07	0.13	0.01
0.250 kHz	2.64	0.39	0.04	0.86	0.11	0.01
0.500 kHz	5.98	2.93	0.86	4.01	2.01	0.61
1.000 kHz	7.42	7.51	9.78	93.97	97.71	136.72
2.000 kHz	22.54	31.00	26.59	64.86	85.74	86.91
4.000 kHz	24.37	31.92	119.30	29.29	37.08	158.74
8.000 kHz	4.68	3.38	8.03	18.11	13.73	36.04
16.000 kHz	2.87	1.00	0.04	7.26	2.38	0.08
155 dB peak SPL	Unwtg.	A-Wtg.	P-Wtg.	Unwtg.	A-Wtg.	P-Wtg.
< 0.125 kHz	438.30	3.35	0.21	27.33	0.15	0.01
0.125 kHz	145.55	2.79	0.18	3.80	0.10	0.01
0.250 kHz	7.16	1.14	0.13	3.28	0.41	0.04
0.500 kHz	11.19	5.72	1.75	17.83	9.65	3.29
1.000 kHz	16.91	17.09	22.18	359.94	369.68	496.32
2.000 kHz	64.88	89.30	74.11	198.26	265.34	250.59
4.000 kHz	69.73	90.79	332.93	92.92	119.44	495.65
8.000 kHz	17.97	14.08	40.77	34.62	27.16	78.23
16.000 kHz	2.95	1.01	0.04	15.74	5.22	0.19
160 dB peak SPL	Unwtg.	A-Wtg.	P-Wtg.	Unwtg.	A-Wtg.	P-Wtg.
< 0.125 kHz	2361.34	15.31	0.98	1242.47	3.87	0.26
0.125 kHz	330.29	6.47	0.43	70.78	1.64	0.11
0.250 kHz	34.38	4.87	0.53	19.41	2.42	0.25
0.500 kHz	60.21	30.30	9.02	229.99	124.46	43.71
1.000 kHz	107.67	103.40	115.54	1911.59	1988.69	2794.80
2.000 kHz	261.16	360.65	293.88	1042.50	1392.56	1329.88
4.000 kHz	205.51	270.11	903.21	264.47	340.09	1309.27
8.000 kHz	70.77	50.54	107.15	191.76	143.65	346.11
16.000 kHz	20.98	7.47	0.34	60.96	18.84	0.57

Table 4. The unweighted and weighted octave band sound exposure levels (dB SEL) for a single reverberant blast wave produced by each of the two sources at the three peak levels.

	Source I			Source III		
150 dB peak SPL						
	Unwtg.	A-Wtg.	P-Wtg.	Unwtg.	A-Wtg.	P-Wtg.
< 0.125 kHz	113.0	91.7	79.7	101.4	80.2	68.2
0.125 kHz	110.6	93.9	82.1	98.9	85.2	73.9
0.250 kHz	98.2	89.9	80.2	93.3	84.4	74.8
0.500 kHz	101.7	98.7	93.3	100.0	97.0	91.9
1.000 kHz	102.7	102.7	103.9	113.7	113.9	115.3
2.000 kHz	107.5	108.9	108.2	112.1	113.3	113.4
4.000 kHz	107.8	109.0	114.7	108.6	109.7	116.0
8.000 kHz	100.7	99.3	103.0	106.6	105.4	109.5
16.000 kHz	98.6	94.0	80.1	102.6	97.7	83.1
155 dB peak SPL						
	Unwtg.	A-Wtg.	P-Wtg.	Unwtg.	A-Wtg.	P-Wtg.
< 0.125 kHz	120.4	99.2	87.3	108.3	85.7	73.7
0.125 kHz	115.6	98.4	86.6	99.8	84.1	72.5
0.250 kHz	102.5	94.6	85.2	99.1	90.1	80.2
0.500 kHz	104.5	101.6	96.4	106.5	103.8	99.1
1.000 kHz	106.3	106.3	107.4	119.5	119.7	120.9
2.000 kHz	112.1	113.5	112.7	117.0	118.2	118.0
4.000 kHz	112.4	113.6	119.2	113.7	114.8	120.9
8.000 kHz	106.5	105.5	110.1	109.4	108.3	112.9
16.000 kHz	98.7	94.0	80.2	106.0	101.2	86.8
160 dB peak SPL						
	Unwtg.	A-Wtg.	P-Wtg.	Unwtg.	A-Wtg.	P-Wtg.
< 0.125 kHz	127.7	105.8	93.9	124.9	99.9	88.1
0.125 kHz	119.2	102.1	90.3	112.5	96.1	84.5
0.250 kHz	109.3	100.9	91.2	106.9	97.8	87.9
0.500 kHz	111.8	108.8	103.5	117.6	114.9	110.4
1.000 kHz	114.3	114.1	114.6	126.8	127.0	128.4
2.000 kHz	118.1	119.6	118.7	124.2	125.4	125.2
4.000 kHz	117.1	118.3	123.5	118.2	119.3	125.1
8.000 kHz	112.5	111.0	114.3	116.8	115.6	119.4
16.000 kHz	107.2	102.7	89.2	111.8	106.7	91.5

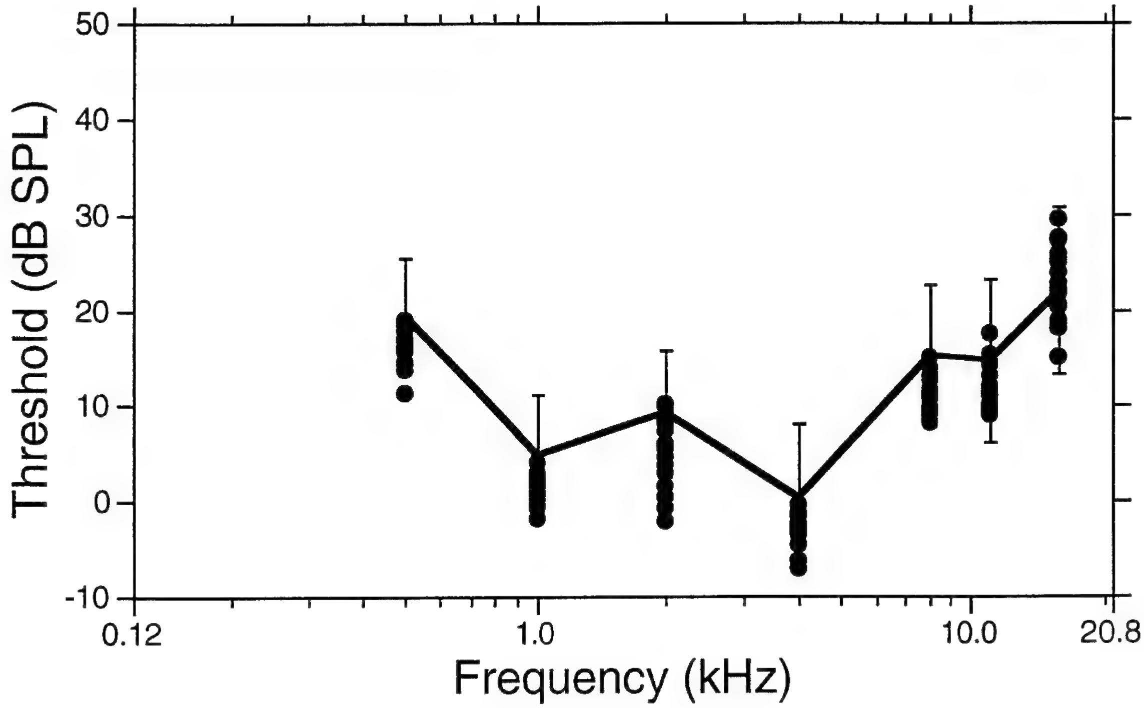

Figure 14. Preexposure AEP thresholds (dB SPL) for the 18 groups of chinchillas exposed to reverberant blast waves (symbols). The solid line represents the mean of 423 normal subjects reported by Hamernik et al. contractor report ADA 241-117 (1991). The bars represent one standard deviation above and below the mean for the normative (laboratory standard) population.

Table 5 Summary of the Analysis of Variance for Preexposure Thresholds for the Nine Groups of Chinchillas Exposed to Reverberant Blast Waves from Source I.

Source of Variation	SS	df	MS	F	p(F)
Groups	2981.15	8	372.64	2.50	.015
Between Groups	18607.45	125	148.86		
Frequency	72392.91	6	12065.49	518.01	.000
Groups by Frequency	3204.30	48	66.76	2.87	.000
Within Groups	17469.01	750	23.29		

Table 6 Summary of the Analysis of Variance for Preexposure Thresholds for the Nine Groups of Chinchillas Exposed to Reverberant Blast Waves from Source III.

Source of Variation	SS	df	MS	F	p(F)
Groups	822.88	8	102.86	.88	.534
Between Groups	14588.28	125	116.71		
Frequency	54293.31	6	9048.88	325.21	.000
Groups by Frequency	1809.84	48	37.71	1.36	.058
Within Groups	20868.43	750	27.82		

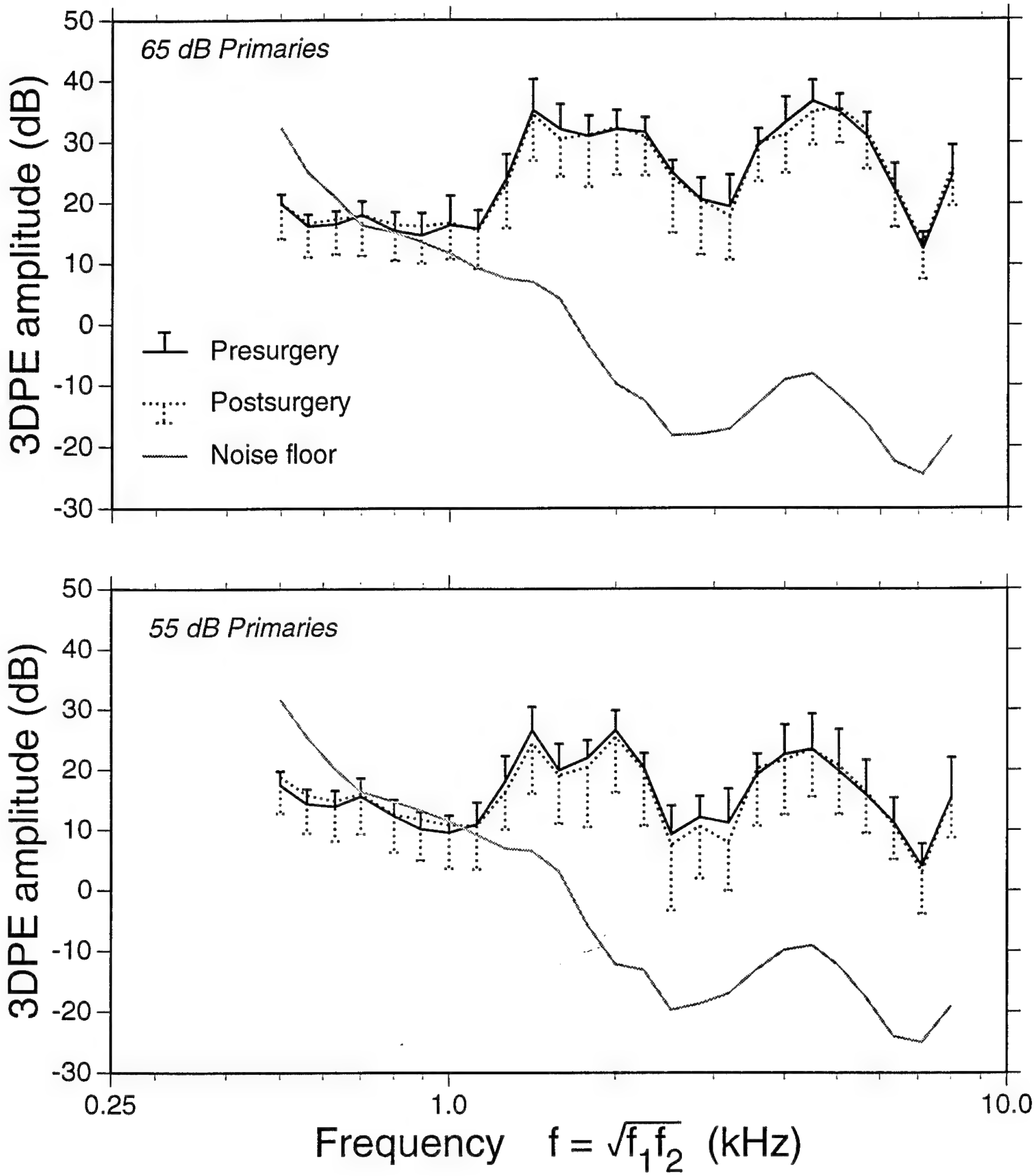

Figure 15. A comparison of the mean DPEgrams for primary tones $L_1 = L_2$ of 55 and 65 dB SPL before and after surgical monauralization and electrode implantation. The presurgical (solid black) line represents the average of nine DPEgrams, one from each chinchilla. The DPEgram from each chinchilla was computed as the mean from 25 sets of DPEgrams measured on 25 different days. The postsurgical (dotted black) line is the average DPEgram from 102 monaural chinchillas. (The gray line represents the noise floor from the monaural chinchillas and is similar to the noise floor of the presurgical group.) Error bars represent one standard deviation.

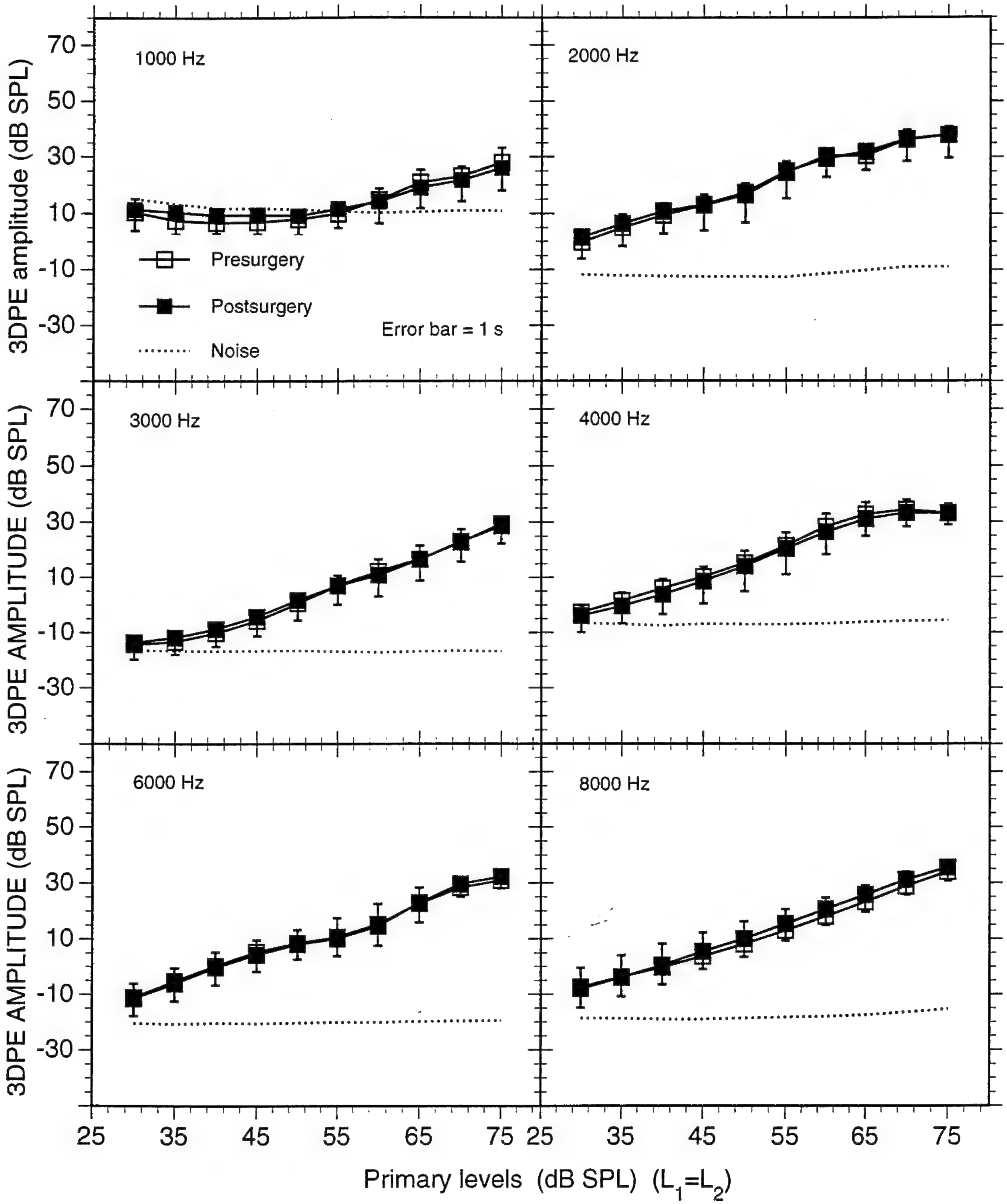

Figure 16. A comparison of the mean input/output (I/O) functions before and after surgical monauralization and electrode implantation. The presurgical line (B) represents the average of nine I/O functions, one from each chinchilla. The I/O function from each chinchilla was computed as the mean from 25 I/O functions measured on 25 different days. The postsurgical (G) is the average I/O function from 104 monaural chinchillas. (The dotted line represents the noise floor from the monaural chinchillas and is similar to the noise of the presurgical group.) Error bars represent one standard deviation.

and the noise floor rapidly decreases. The I/O functions shown in Figure 16 are very similar to those reported by Subramanian et al. (1994) using similar equipment except for the 1.0 kHz data. At this frequency our noise floor is considerably higher and reliable 3DPEs could not be measured with primaries below about 70 dB SPL. Thresholds at 1.0 kHz for the Subramanian et al. data are about 35 dB SPL. Since the noise levels generally increased at the low frequencies the differences in the noise floors between our results and those of Subramanian et al. may be the result of differences in averaging times and methods of averaging. Figure 17 shows the mean preexposure DPEgrams at 55 and 65 dB SPL plotted together along with the corresponding 3DPE data taken from the I/O functions. As expected the DPEgram and I/O data acquisition protocols produce consistent results.

When the 3DPEs were collected on normal binaural animals (N = 9) there were virtually no differences in the mean data as seen in Figures 15 and 16 where DPEgrams and I/O functions from the binaural animals are compared with the respective normative data base acquired on monaural animals. A further indication of test-retest reliability can be obtained from Tables 7 through 10 in which the standard deviations of the 3DPE data are compared for each animal before and after surgery. (Note: In the postsurgery group, Subjects 1771 and 1774 developed complications and could not be tested after recovery from surgery.) Tables 7 and 8 list the standard deviations of the presurgery data pool for which 55 dB SPL DPEgrams were measured over 25 days and of the postsurgery data pool taken over 5 days of testing respectively. Tables 9 and 10 provide similar results for 65 dB SPL DPEgrams. Reading down the columns provides an indication of the average standard deviation of the 3DPE measurement for a given animal across all frequencies tested, while reading across each row, provides an indication of the average standard deviation across animals for a given test frequency. In general, for both the 55 and 65 dB SPL DPEgrams average standard deviations are less than 10 dB.

D. Postexposure AEP Thresholds. AEP threshold recovery functions were measured over a 30-day postexposure period at the 0.5, 2.0 and 8.0 kHz test frequencies following the 18 different blast wave exposures. These recovery functions are shown in Figures 18 and 19 for Source I and III respectively. Visual inspection of these two figures shows the effect of increasing the number of impulses at a given intensity by scanning across the rows or the effect of intensity for a given number of impulses by scanning down the columns. Comparing like cells in Figures 18 and 19 shows the effect of spectrum, all other parameters being the same. In general, there are clear differences in effects on thresholds during recovery for the two sources. Source III for a given set of parameters typically produces greater losses than do the Source I blast waves. For each source increasing the number or peak SPL of the impulses, as expected, increases the threshold shifts. Also evident is the relatively slow or delayed threshold recovery once threshold shifts (TS) exceed about 40 dB as is the growth of TS over time for the more severe exposure conditions. This delayed recovery or growth of TS phenomena has been described by Hamernik et al. (1988) and was shown to be associated with permanent hearing loss and cochlear lesions.

Figures 20 and 21 provide another perspective on the early postexposure threshold shifts. In these figures the maximum TS (TS_{max}), regardless of when it occurs, is shown as a function of frequency for the 18 exposures. The same commentary is valid for TS_{max} behavior

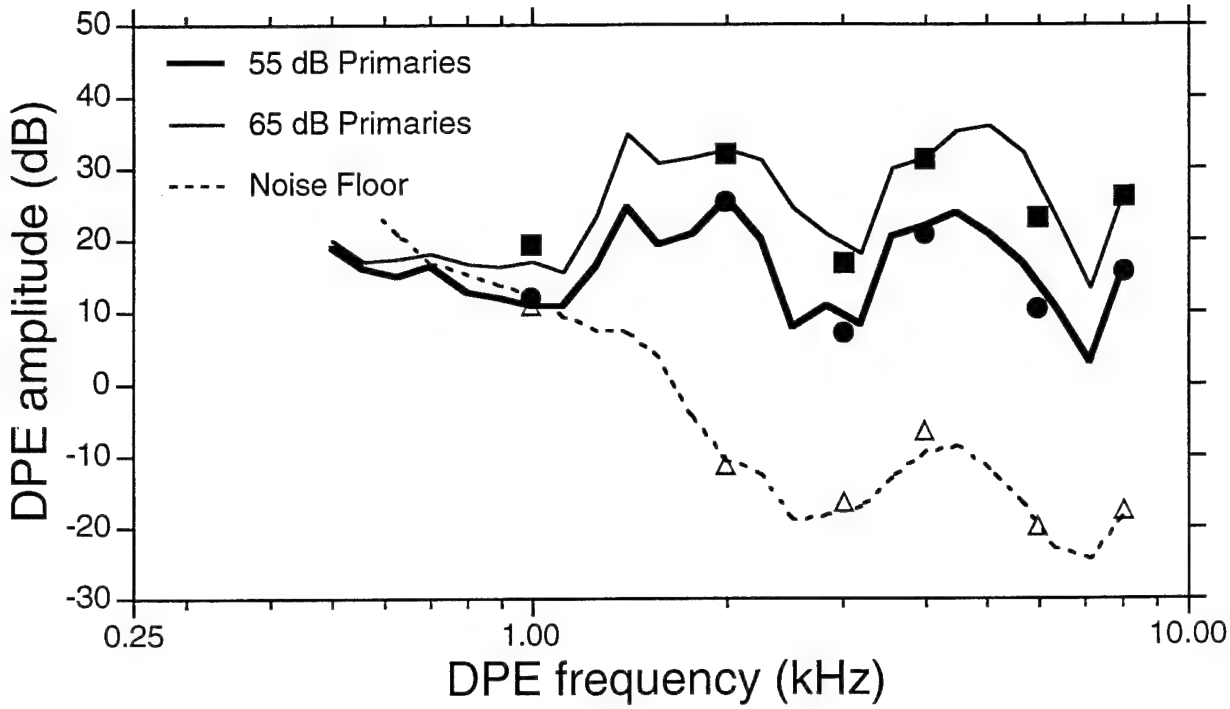

Figure 17. The mean cubic distortion product otoacoustic emission (3DPE) as a function of frequency (DPEgram) collected on 102 non-exposed, monaural chinchillas using the Virtual Model M330 Otoacoustic Emissions Test Instrument. The dashed line on the graph is the average of the mean noise floors from both of the primary curves. The closed symbols represent the level of the 3DPE obtained at 55 or 65 dB during the collection of input-output functions. The open triangles represent the average of the noise floors at 55 and 65 dB during collection of the 3DPE input-output functions.

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across the 18 exposure conditions as was made for the TS recovery functions. The threshold dynamics in the 30-day postexposure period were consistent and systematic in showing increasing losses of sensitivity as number of impulses and impulse levels were increased and as the spectrum shifted to the higher frequencies present in the Source III wave forms.

The permanent threshold shift (PTS) audiograms following each of the 18 exposure conditions are shown in Figures 22 and 23. As with TS dynamics, PTS showed an orderly progression of loss across test frequencies as the energy in the exposure was increased. In general for all the 1X exposures, there was no PTS. For the impulses produced by Source I which are dominated by low frequency energy the most severe PTS did not exceed about 30 dB, while for the Source III impulses with more energy in the mid frequencies, mean PTS exceeded 40 dB at some frequencies. When a PTS was observed, a broadband loss was typically seen, extending across the entire range of test frequencies.

Table 7. The standard deviations of the presurgery 3DPE data that formed the 55 dB SPL DPEgram shown in Figure 15. The standard deviation for each animal at each frequency was computed from the 75 DPEgrams measured over 25 days. The final row and column represents the average standard deviation for that respective row or column.

Frequency (kHz)	Subject Number								
	1766	1767	1769	1770	1771	1772	1773	1774	1775	Mean
0.500	9.56	9.24	8.95	8.17	7.46	8.52	8.74	10.30	8.31	8.81
0.560	10.04	9.19	9.41	9.60	6.96	7.51	8.55	9.00	7.20	8.61
0.630	11.31	9.93	11.14	8.34	8.65	8.47	7.06	9.08	7.80	9.09
0.700	11.78	9.80	11.03	9.28	9.06	8.88	9.43	7.39	8.14	9.42
0.800	10.79	8.52	9.56	6.69	7.92	8.59	9.18	7.88	7.27	8.49
0.890	8.83	9.63	8.86	7.32	7.98	6.21	7.99	6.10	5.29	7.58
1.000	9.76	8.54	11.12	6.74	7.77	5.44	7.49	6.91	6.30	7.79
1.120	8.03	7.95	9.84	7.17	5.93	5.27	8.50	5.08	3.41	6.80
1.260	5.72	5.63	7.34	6.27	5.69	4.64	7.65	4.77	3.23	5.66
1.410	8.65	8.75	9.28	5.68	7.40	7.92	13.29	8.02	9.91	8.77
1.580	10.44	9.57	8.75	4.17	7.93	7.05	10.70	6.62	6.83	8.01
1.780	10.27	9.07	11.17	13.49	13.36	12.65	11.40	11.26	11.14	11.53
2.000	10.79	7.50	9.55	12.59	11.24	10.85	9.39	9.45	8.45	9.98
2.250	9.73	7.37	10.38	10.90	8.76	11.66	13.65	8.22	8.80	9.94
2.520	10.50	12.50	7.93	9.75	9.86	9.26	11.66	8.98	12.71	10.35
2.830	10.60	5.15	8.96	11.58	7.41	11.82	10.96	4.70	8.43	8.85
3.180	7.61	7.16	7.02	5.99	7.51	11.20	10.96	6.52	8.20	8.02
3.570	9.27	5.86	11.47	9.93	10.88	9.82	12.40	10.52	10.53	10.08
4.000	8.76	5.46	10.19	11.63	9.53	10.40	9.60	9.01	9.41	9.33
4.490	7.34	4.49	8.47	9.24	7.65	8.26	9.50	7.40	8.55	7.88
5.040	5.83	5.39	7.22	8.53	5.97	8.32	11.48	8.48	6.58	7.53
5.660	4.49	5.17	4.75	4.67	3.24	8.44	10.47	5.43	7.44	6.01
6.350	9.31	5.40	6.20	6.63	4.77	5.29	10.07	3.24	3.99	6.10
7.120	5.96	5.91	8.62	7.15	5.97	5.21	8.07	4.38	5.69	6.33
8.000	5.68	6.09	8.54	8.56	7.54	6.75	8.91	5.34	4.97	6.93
Mean	8.84	7.57	9.03	8.40	7.86	8.34	9.88	7.36	7.54	8.31

Table 8. The standard deviations of the postsurgery 3DPE data obtained from the animals whose presurgery data are shown in Table 7. The standard deviation for each animal at each frequency was computed from the 15 DPEgrams measured over 5 days. The final row and column represents the average standard deviation for that respective row or column.

Frequency (kHz)	Subject Number							
	1766	1767	1769	1770	1772	1773	1775	Mean
0.500	5.52	7.99	5.57	8.74	10.93	5.30	7.64	7.39
0.560	8.90	9.50	7.75	10.04	6.81	7.70	6.68	8.20
0.630	9.15	8.77	5.96	7.43	4.69	5.76	5.71	6.78
0.700	9.67	5.69	7.39	6.35	6.12	8.59	7.01	7.26
0.800	9.65	5.26	6.79	6.46	6.20	10.26	5.85	7.21
0.890	10.74	5.47	8.54	10.52	6.30	5.90	6.78	7.75
1.000	7.32	4.84	10.52	4.18	2.52	9.57	4.99	6.28
1.120	6.19	7.01	11.54	7.12	4.07	7.70	4.79	6.92
1.260	5.04	4.86	4.83	4.84	2.19	7.22	3.07	4.58
1.410	3.56	4.06	6.53	2.87	2.85	4.14	4.87	4.13
1.580	2.27	5.11	7.38	3.42	3.29	5.13	3.18	4.26
1.780	3.23	2.87	2.49	3.28	6.80	7.45	9.29	5.06
2.000	2.63	3.68	4.10	7.19	7.62	8.72	7.66	5.94
2.250	5.89	5.34	5.78	1.40	4.84	5.30	8.57	5.30
2.520	3.62	2.09	3.20	2.68	2.41	4.63	11.06	4.24
2.830	2.67	2.76	2.80	2.69	3.25	3.19	3.11	2.92
3.180	3.25	2.63	3.42	2.82	1.79	5.04	4.27	3.32
3.570	2.19	2.20	3.52	3.23	1.67	4.94	3.33	3.01
4.000	3.73	4.09	2.75	3.08	3.31	6.00	2.72	3.67
4.490	3.70	5.64	4.15	4.90	5.42	7.27	2.66	4.82
5.040	3.36	4.88	5.86	4.90	3.87	5.19	4.87	4.70
5.660	2.46	5.25	4.35	3.22	2.02	4.06	7.00	4.05
6.350	2.85	4.41	6.53	3.43	3.93	3.83	6.77	4.54
7.120	7.44	8.21	11.64	12.69	8.81	4.79	3.01	8.08
8.000	6.30	3.36	8.88	9.37	5.92	3.48	3.17	5.78
Mean	5.25	5.04	6.09	5.47	4.71	6.05	5.52	5.45

Table 9. The standard deviations of the presurgery 3DPE data that formed the 65 dB SPL DPEgram shown in Figure 15. The standard deviation for each animal at each frequency was computed from the 75 DPEgrams measured over 25 days. The final row and column represents the average standard deviation for that respective row or column.

Frequency (kHz)	Subject Number								
	1766	1767	1769	1770	1771	1772	1773	1774	1775	Mean
0.500	9.54	10.09	9.71	9.47	8.27	6.96	9.52	9.23	9.38	9.13
0.560	9.24	10.34	10.64	9.75	7.92	6.85	11.06	8.22	8.45	9.16
0.630	11.83	9.80	8.69	8.88	7.72	6.90	9.60	9.40	6.25	8.79
0.700	11.35	8.75	9.30	9.29	8.40	6.95	8.39	7.35	7.45	8.58
0.800	6.76	6.62	9.15	7.68	7.48	5.58	7.83	8.13	5.00	7.14
0.890	6.20	6.24	7.49	6.48	6.78	4.05	6.48	6.69	4.23	6.07
1.000	7.63	5.19	7.94	6.62	4.74	3.77	6.70	5.45	4.03	5.78
1.120	7.51	6.88	8.19	5.53	6.21	4.24	7.28	4.44	4.51	6.09
1.260	7.76	6.95	6.58	3.77	6.78	7.59	11.88	5.52	7.33	7.13
1.410	9.33	6.40	9.21	6.88	9.16	6.07	12.31	6.79	6.11	8.03
1.580	8.02	4.32	7.95	6.64	5.31	4.83	10.60	4.15	3.16	6.11
1.780	12.50	9.58	9.75	8.58	7.62	5.65	10.51	8.45	7.82	8.94
2.000	10.35	7.93	7.72	8.14	7.10	7.37	7.62	8.33	7.12	7.96
2.250	9.14	6.54	8.26	6.56	5.37	8.09	12.53	5.31	4.15	7.33
2.520	10.31	9.70	9.12	8.26	9.51	4.73	12.67	8.94	9.25	9.16
2.830	9.79	5.09	9.90	10.76	10.27	7.68	11.93	7.04	7.49	8.88
3.180	8.10	7.30	6.89	4.78	8.95	6.28	13.55	7.95	9.15	8.11
3.570	8.31	5.11	7.62	7.33	6.15	4.60	13.59	4.97	4.49	6.91
4.000	7.81	5.37	6.09	7.30	4.68	5.01	9.58	5.37	4.41	6.18
4.490	7.73	4.71	6.56	7.75	5.68	5.32	11.24	5.28	4.57	6.54
5.040	8.24	4.60	6.75	8.33	5.19	7.03	12.44	4.77	4.66	6.89
5.660	8.49	4.22	10.91	11.88	5.40	6.86	12.75	4.38	5.93	7.87
6.350	8.15	6.60	10.41	9.12	7.22	7.33	14.22	6.11	6.65	8.42
7.120	5.74	5.93	8.16	8.03	3.36	5.38	10.38	5.22	6.93	6.57
8.000	7.02	6.99	9.66	9.31	5.48	7.11	11.99	5.34	6.46	7.71
Mean	8.67	6.85	8.51	7.88	6.83	6.09	10.67	6.51	6.20	7.58

Table 10. The standard deviations of the postsurgery 3DPE data obtained from the animals whose presurgery data are shown in Table 9. The standard deviation for each animal at each frequency was computed from the 15 DPEgrams measured over 5 days. The final row and column represents the average standard deviation for that respective row or column.

Frequency (kHz)	Subject Number							
	1766	1767	1769	1770	1772	1773	1775	Mean
0.500	7.62	7.08	5.63	6.03	4.59	6.69	8.48	6.59
0.560	8.75	9.72	7.82	7.86	3.70	6.24	6.32	7.20
0.630	7.96	7.28	5.88	7.80	3.63	4.19	5.64	6.05
0.700	9.46	7.70	9.29	6.98	4.78	6.07	8.02	7.47
0.800	8.12	6.16	5.71	6.96	3.73	4.32	4.66	5.67
0.890	7.43	7.74	6.48	6.48	3.36	5.87	6.32	6.24
1.000	5.67	7.20	8.91	5.15	2.69	6.40	3.93	5.71
1.120	7.18	4.60	5.80	3.89	4.48	6.80	6.50	5.61
1.260	3.52	6.88	4.73	2.56	4.42	6.48	4.07	4.66
1.410	5.36	5.74	8.85	4.06	8.09	9.09	2.62	6.26
1.580	5.61	5.06	3.38	5.72	3.82	4.00	3.78	4.48
1.780	6.07	4.17	5.45	3.29	3.81	4.55	3.62	4.42
2.000	3.47	4.24	7.06	4.55	2.36	3.28	4.29	4.18
2.250	1.34	2.15	3.71	4.22	3.54	5.34	5.34	3.66
2.520	2.59	2.67	7.12	5.64	5.44	7.62	9.79	5.84
2.830	2.59	2.32	6.44	7.69	5.96	4.03	6.70	5.11
3.180	6.20	4.10	2.90	3.94	4.79	6.61	7.13	5.10
3.570	4.27	3.84	3.12	2.74	3.16	6.65	8.88	4.66
4.000	4.18	5.15	3.58	2.26	3.81	4.53	5.79	4.19
4.490	2.88	5.66	2.62	3.50	4.87	3.86	5.95	4.19
5.040	2.56	4.40	7.41	5.22	5.39	6.08	6.10	5.31
5.660	6.32	3.54	7.45	7.12	7.87	8.43	5.60	6.62
6.350	9.95	2.77	10.82	7.47	10.49	12.02	6.01	8.50
7.120	7.12	9.03	11.19	9.51	13.01	7.38	7.00	9.18
8.000	7.14	11.44	7.38	9.52	8.57	3.98	10.10	8.31
Mean	5.73	5.62	6.35	5.61	5.21	6.02	6.11	5.81

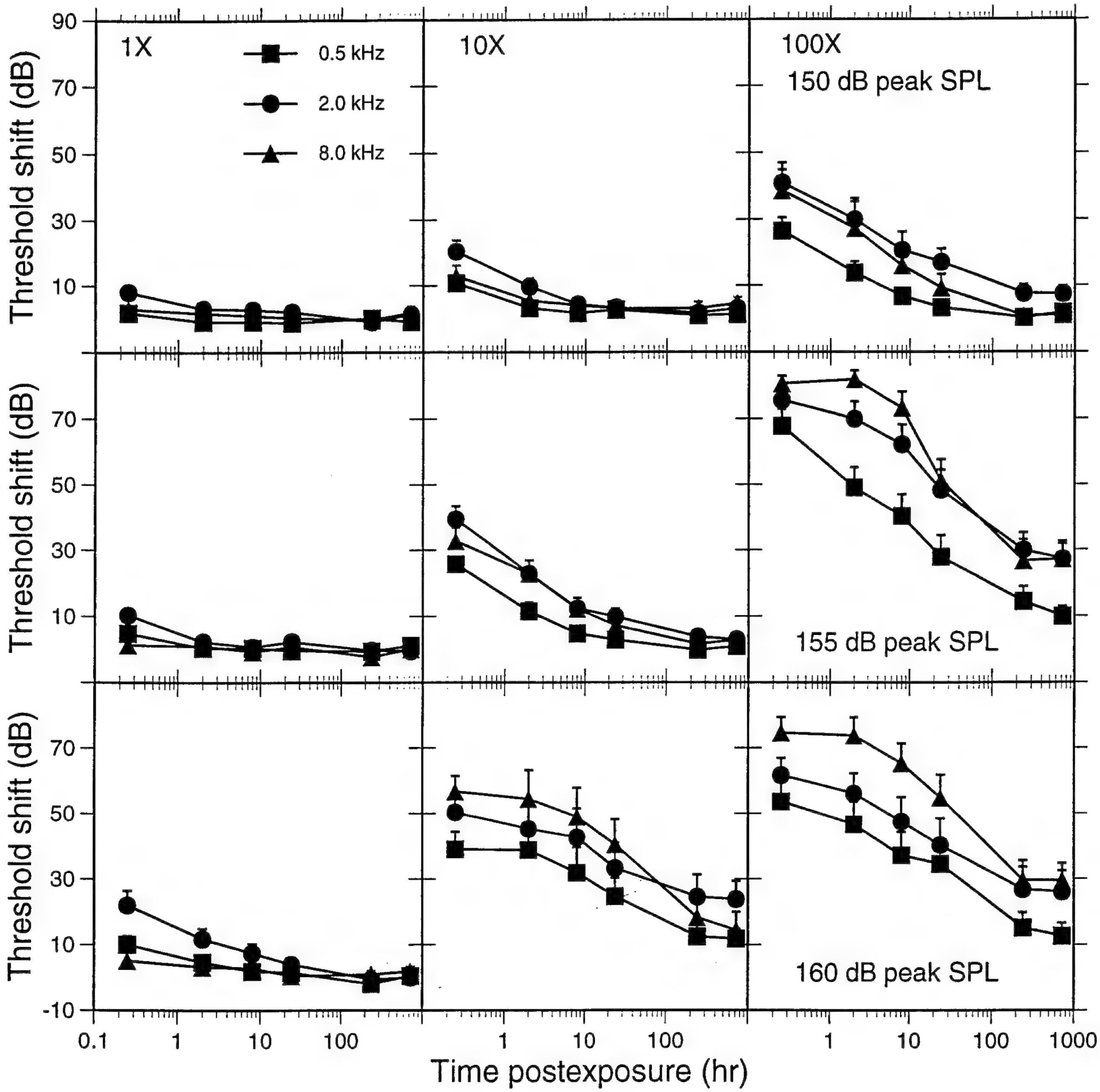

Figure 18. Group mean threshold shift recovery functions at 0.5 kHz, 2.0 kHz, and 8.0 kHz for groups of animals exposed to 1 (left panels), 10 (center panels), or 100 (right panels) reverberant impulses from Source I. The peak intensity of the impulse exposure was 150 dB (top panels), 155 dB (middle panels), or 160 dB (bottom panels). Error bars represent one standard error of the mean.

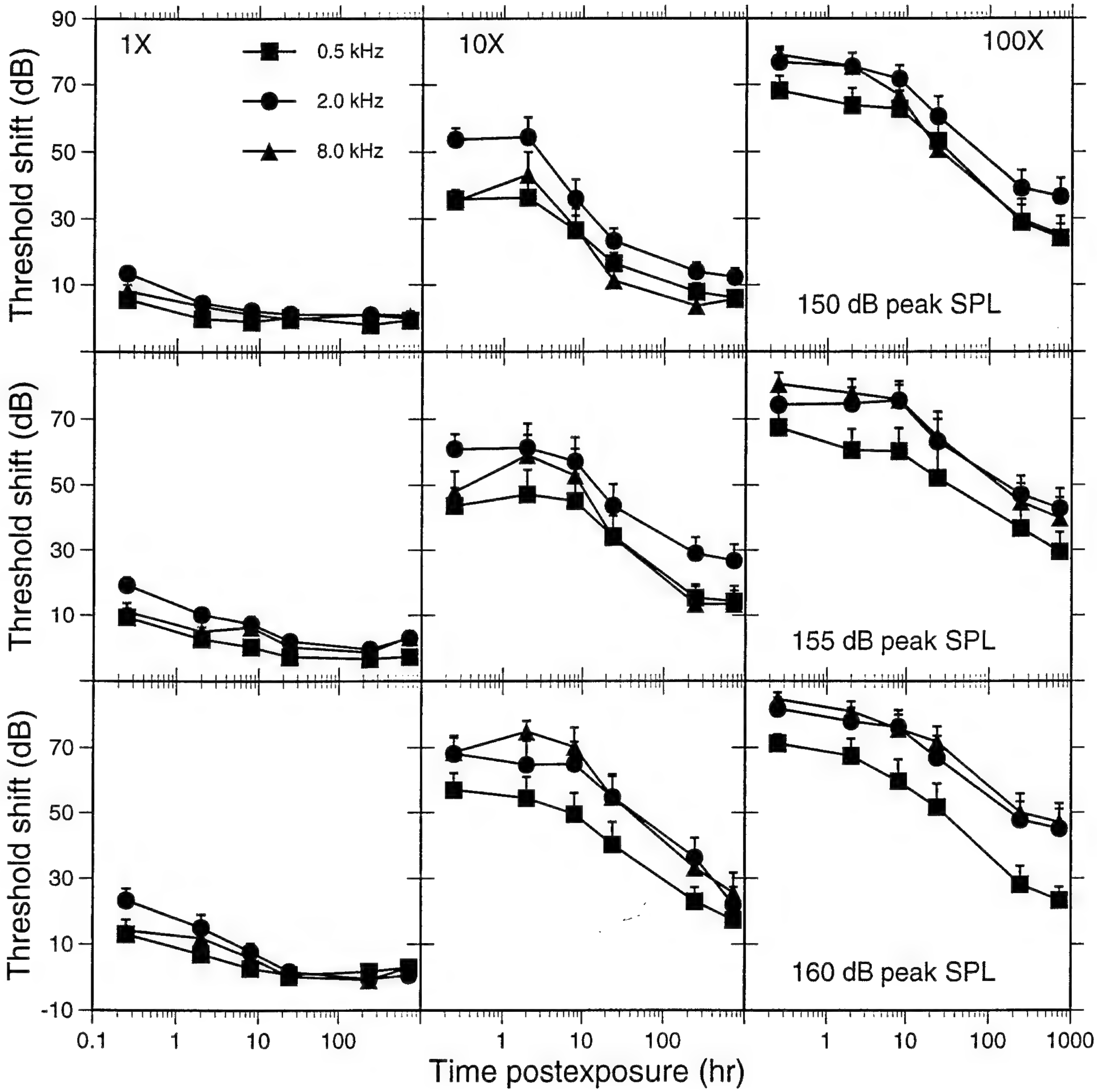

Figure 19. Group mean threshold shift recovery functions at 0.5 kHz, 2.0 kHz, and 8.0 kHz for groups of animals exposed to 1 (left panels), 10 (center panels), or 100 (right panels) reverberant impulses from Source III. The peak intensity of the impulse exposure was 150 dB (top panels), 155 dB (middle panels), or 160 dB (bottom panels). Error bars represent one standard error of the mean.

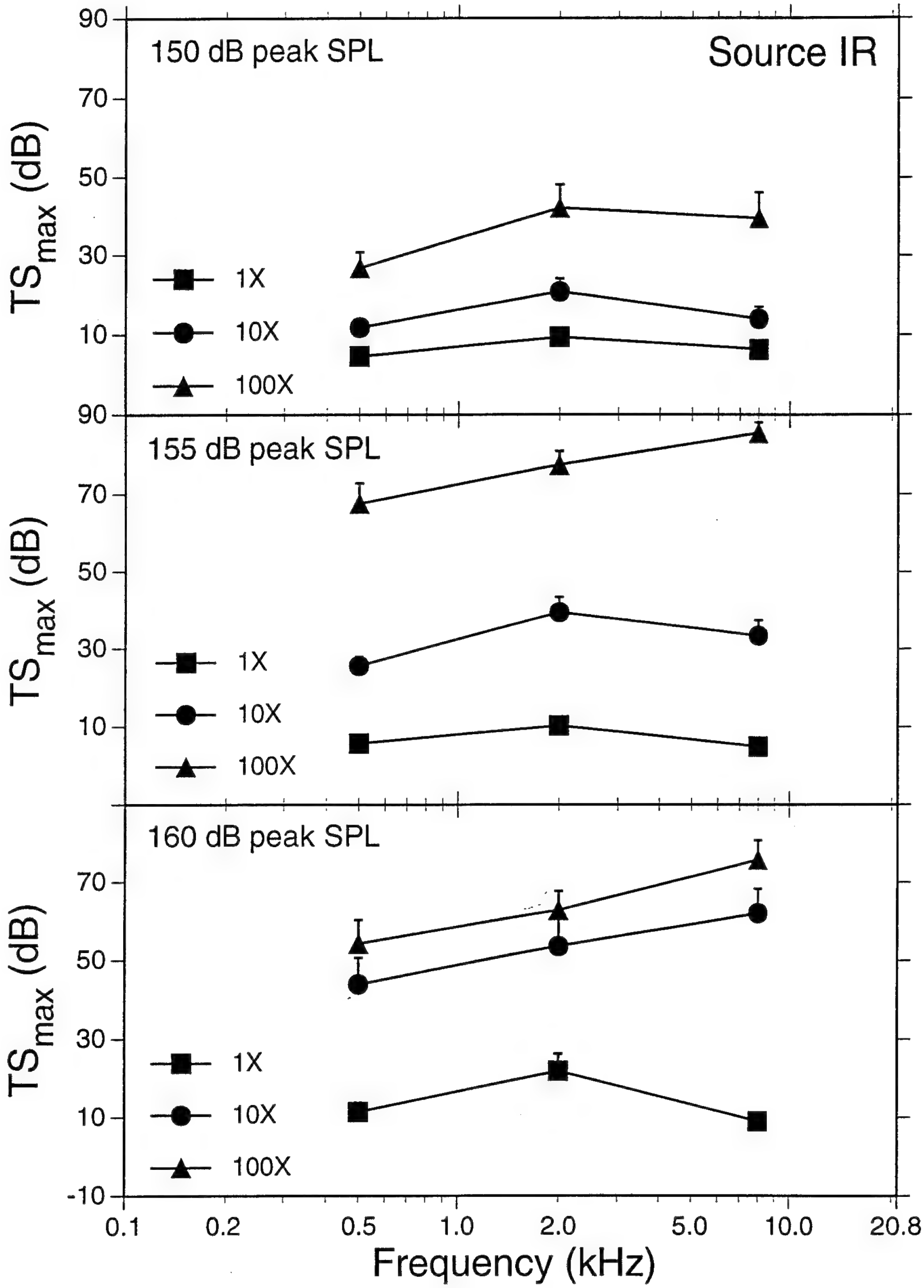

Figure 20. The group mean maximum threshold shift (TS_{max}) at the three test frequencies following exposure to reverberant blast waves produced by Source I at each of the nine conditions indicated. Error bars represent one standard error of the mean.

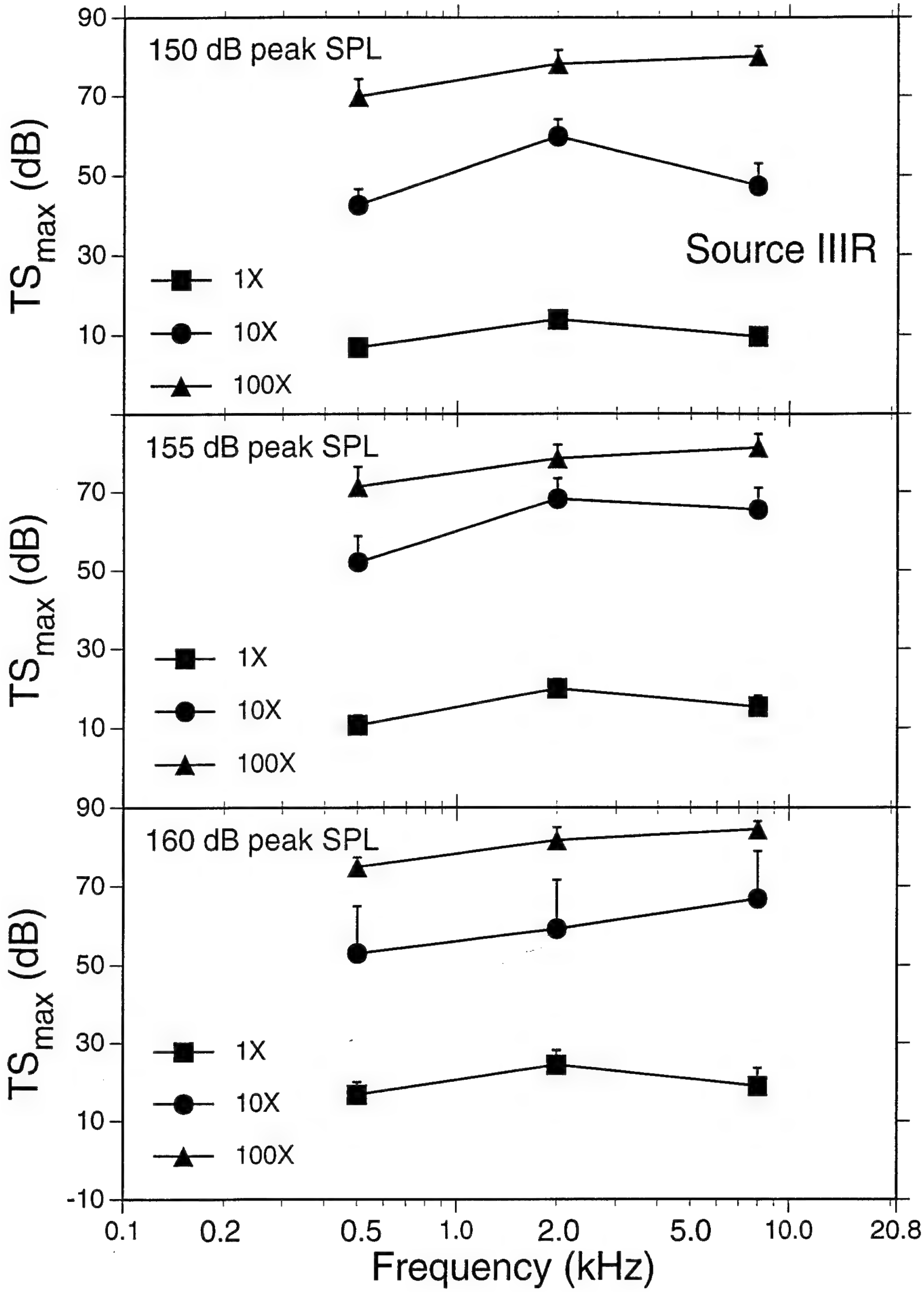

Figure 21. The group mean maximum threshold shift (TS_{max}) at the three test frequencies following exposure to reverberant blast waves produced by Source III at each of the nine conditions indicated. Error bars represent one standard error of the mean.

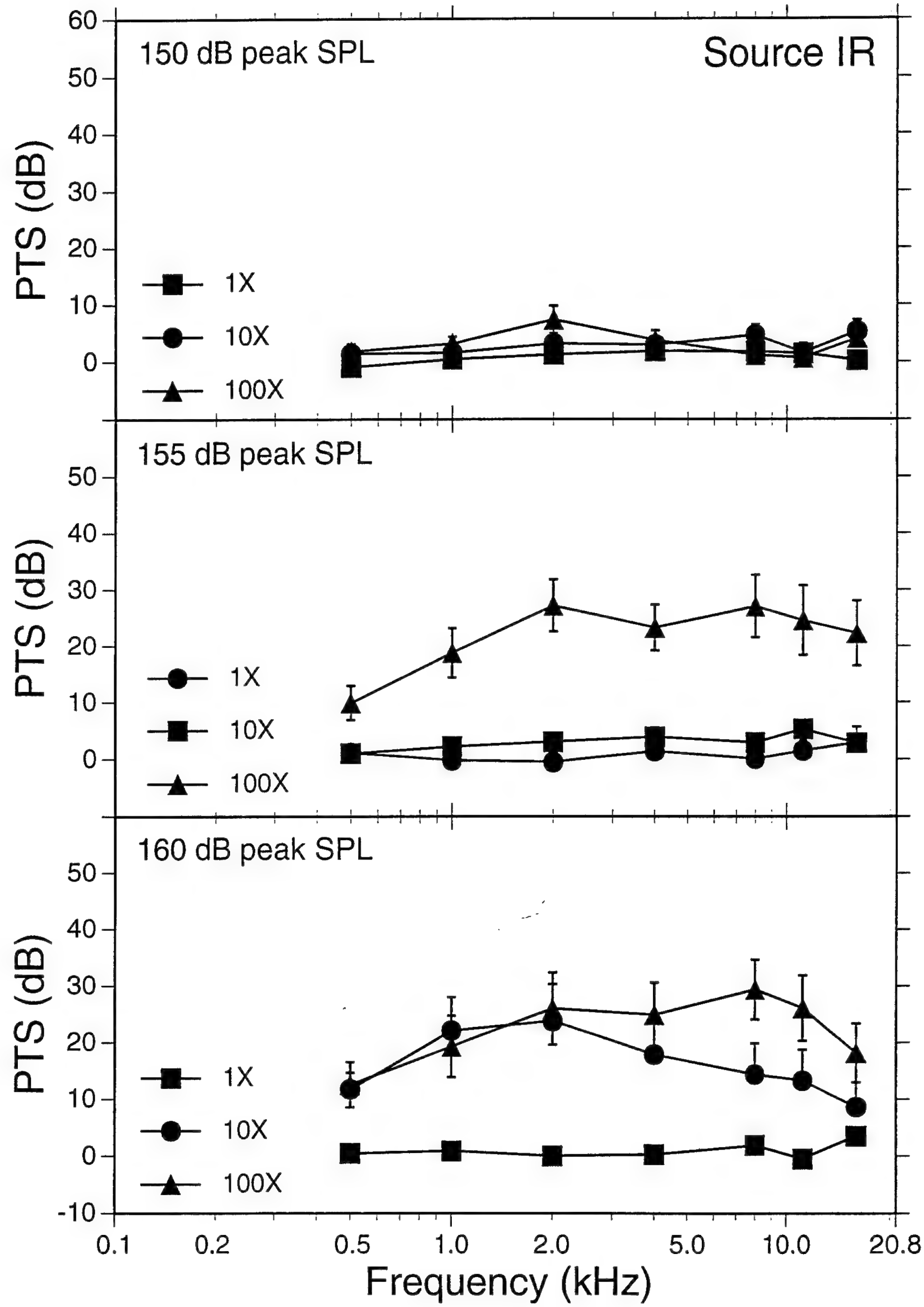

Figure 22. The group mean permanent threshold shift (PTS) audiograms measured 30 days after exposure to reverberant blast waves produced by Source I at each of the nine conditions indicated. Error bars represent one standard error of the mean.

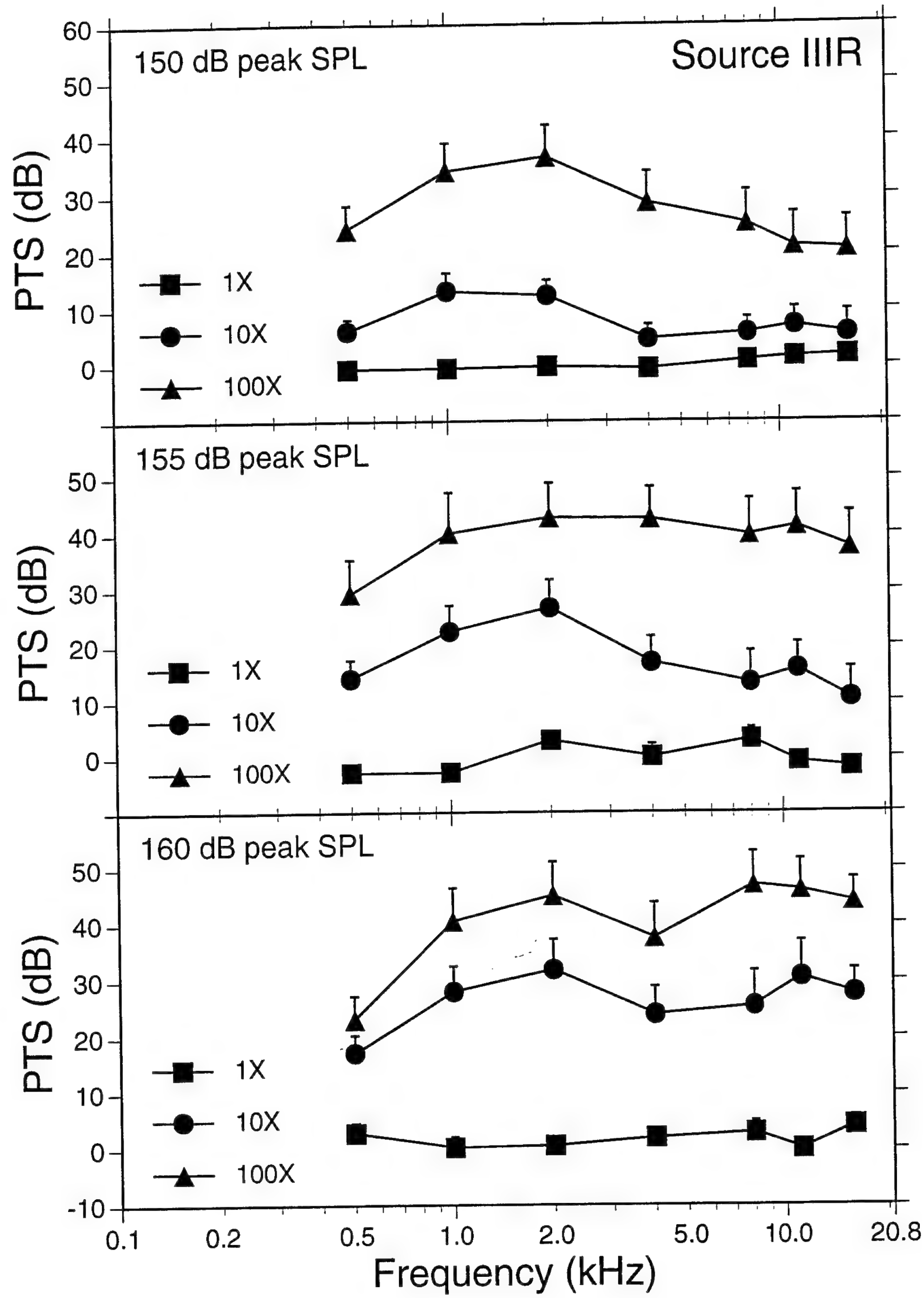

Figure 23. The group mean permanent threshold shift (PTS) audiograms measured 30 days after exposure to reverberant blast waves produced by Source III at each of the nine conditions indicated. Error bars represent one standard error of the mean.

E. Postexposure 3DPE Functions - DPEgrams and I/O Functions. As explained above, 3DPE functions were not acquired systematically on a very large number of animals or for a large number of exposure conditions and the postexposure data should be considered preliminary. In the figures discussed in this section, only data for which reliable data acquisition protocols were employed are reported. Although the postexposure data are limited to Source I exposures they do give an indication of the manner in which PTS and 3DPEs covary. For the 150 dB peak SPL Source I exposures, Figures 24 through 27 show that, as with the PTS shown in Figure 22, 3DPEs did not change appreciably following any of the three exposure conditions. At the 155 dB peak SPL exposures, appreciable changes in the 3DPE functions were seen only in the group exposed to 100 impulses as seen in Figures 28 through 31. 3DPE shifts on the order of 10-20 dB were seen in the DPEgrams across the 1.0 to 8.0 kHz region (Figure 28) and comparable changes were measured in the I/O functions (Figure 31). Although there was no PTS for the 1X and 10X, 155 dB Source I exposure, the I/O function at 2.0 kHz for the 1X exposure was slightly, but consistently depressed and the I/O functions at 2.0, 3.0, and 4.0 kHz, the area usually first affected by the impulses, were consistently depressed for the 10X exposure relative to the normative I/O functions. At the highest exposure levels, 160 dB peak SPL, similar shifts in DPEgrams and I/O functions were recorded (Figures 32 through 35) at both the 10X and 100X conditions across a broad range of frequencies. The 1X exposure conditions showed no changes in 3DPEs just as with the PTS metrics (Figure 22). While direct comparisons between PTS data of Figure 22 and the 3DPE data in Figures 24 through 35 have not been made because of the preliminary nature of the 3DPE data and the unavoidable selection effects inherent in the different sample sizes, it should be noted that there are parallel shifts in the two metrics whenever the exposure showed an effect.

F. Histological Assessment. The pattern of group mean sensory cell losses for the 18 exposure conditions are presented in Figures 36 through 39, grouped according to blast wave source. These four figures present the group mean outer hair cell (OHC) losses (Figures 36 and 38) and inner hair cell (IHC) losses (Figures 37 and 39) within octave-band lengths of the basilar membrane defined by the center frequencies shown. As with the PTS data, there is an order to the pattern of sensory cell loss; damage increases with increasing energy of exposure determined by either peak SPL or number of impulses. For a given exposure energy, Source III produces more sensory cell loss than does Source I, thus highlighting the role of the energy spectrum in producing trauma. In general, it would appear that the lesions develop in the 1.0 to 4.0 kHz region of the cochlea and with increasing exposure energy, spread to the higher frequencies more than to the low such that in the most severe cases there can be a nearly complete OHC loss basalward of the 1.0 kHz region of the cochlea. IHC losses are generally much less than OHC losses and tend to be focused in the 1.0 to 4.0 kHz region of the cochlea.

G. Data Summary. An overview of the permanent effects on hearing and sensory cell populations for each exposure condition is presented in Figures 40 through 45. Each figure shows the AEP audiometric results (PTS) in conjunction with sensory cell losses for a given peak SPL and source as the number of impulses is increased. There is an overall congruence between the audiometric and histological data and both sets of metrics are ordered in increasing severity of effect with the increasing energy in the exposure stimulus.

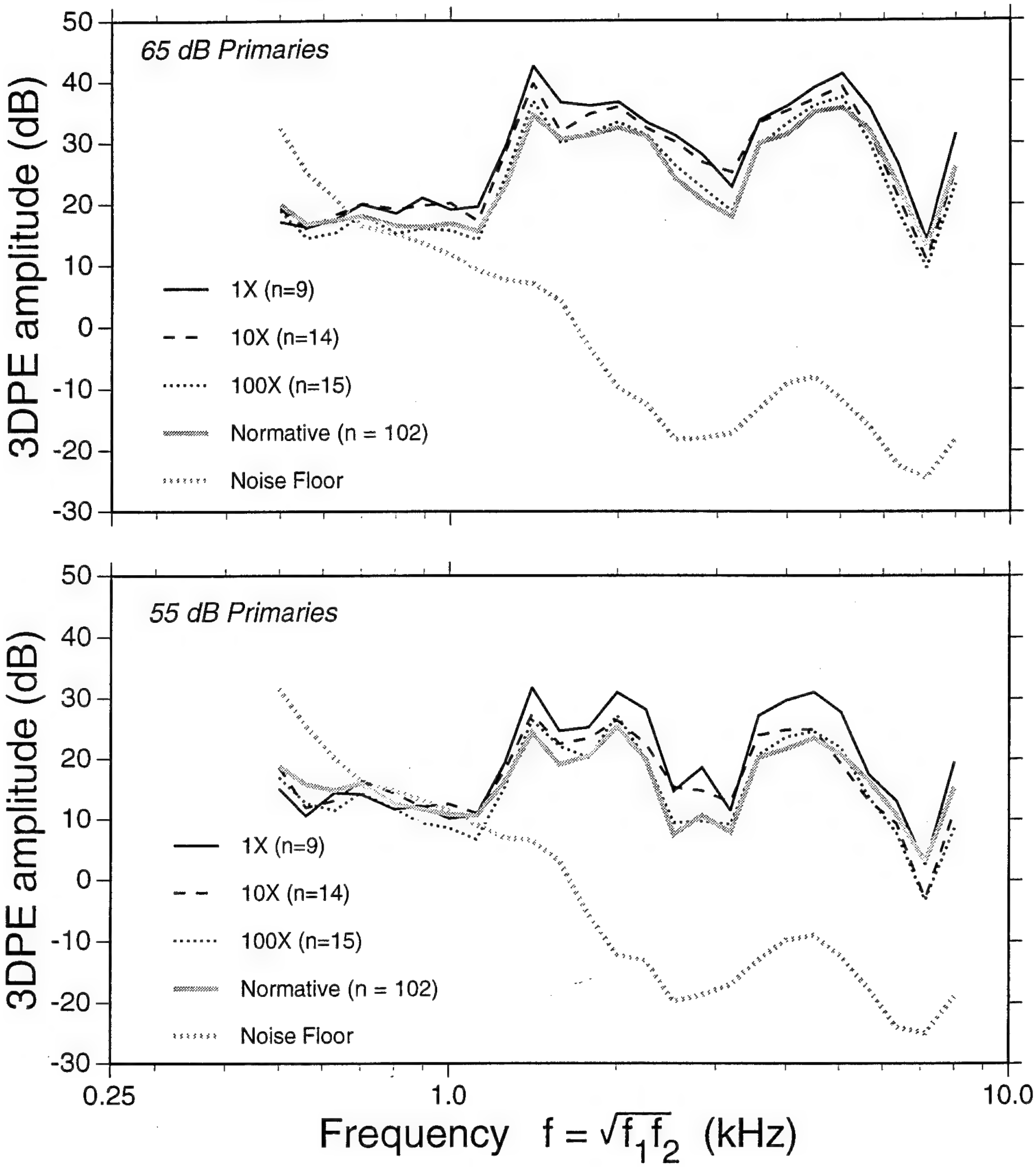

Figure 24. The group mean DPEgrams from three groups of chinchillas recorded at least 30 days after being exposed to 1, 10, or 100 reverberant blast waves from Source I at 150 dB peak SPL. The solid gray line represents the DPEgram from a normative group of 102 monaural chinchillas. The dotted gray line represents the noise floor from the normative sample.

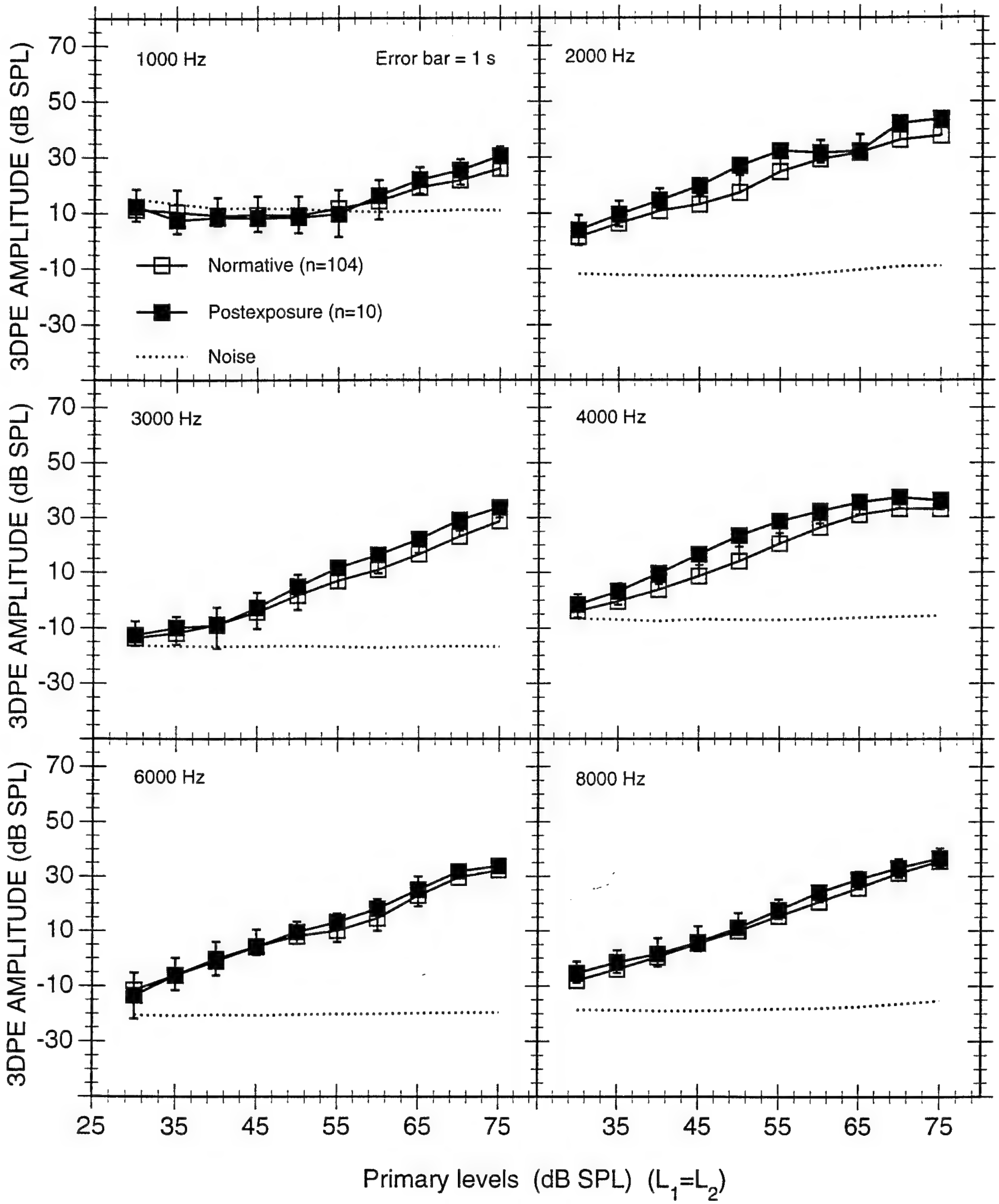

Figure 25. The mean postexposure (150 dB peak SPL, 1X, Source I) input/output functions at the indicated test frequencies ($f = \sqrt{f_1 f_2}$) compared with the mean normative functions. Error bars represent one standard deviation.

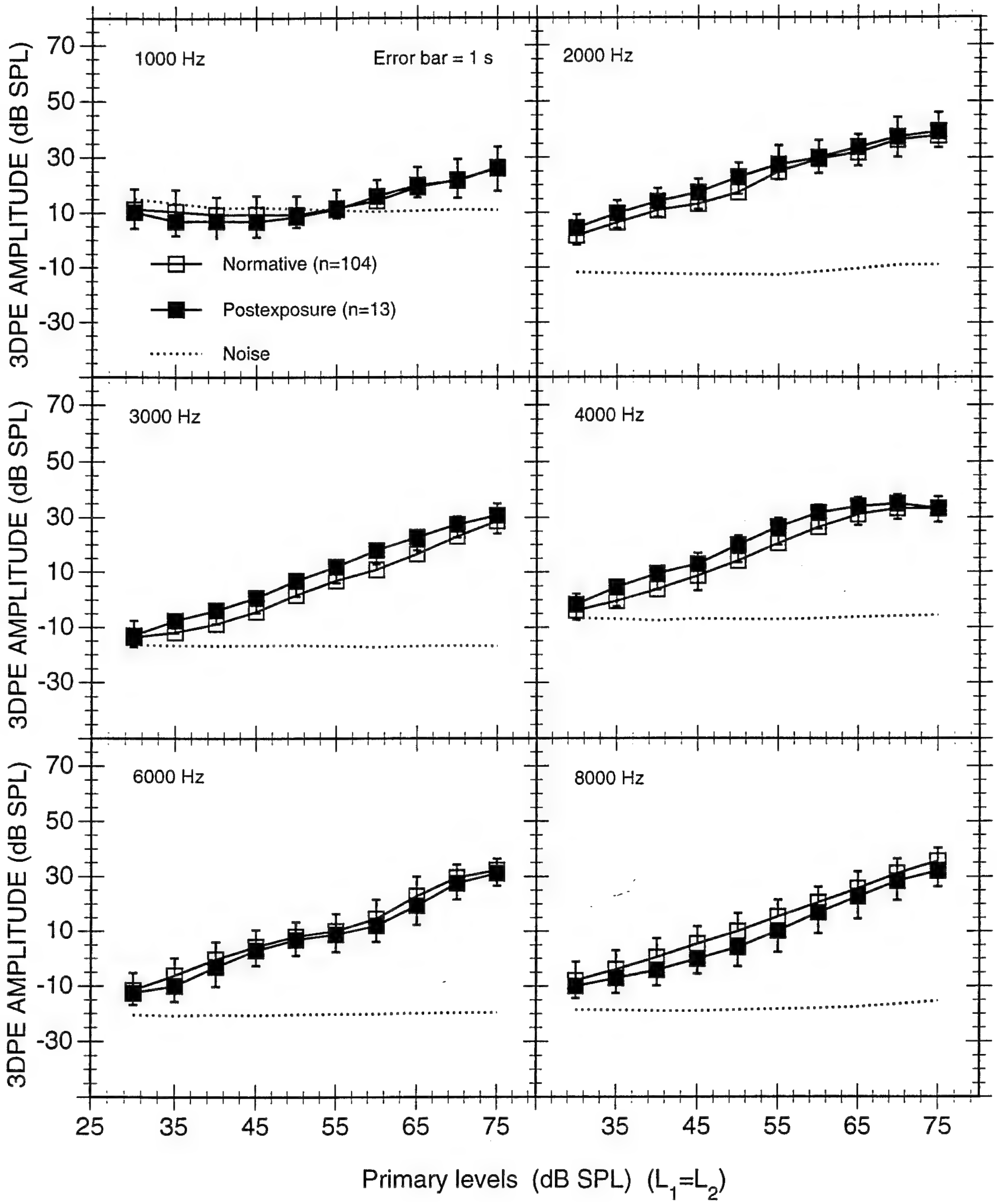

Figure 26. The mean postexposure (150 dB peak SPL, 10X, Source I) input/output functions at the indicated test frequencies ($f = \sqrt{f_1 f_2}$) compared with the mean normative functions. Error bars represent one standard deviation.

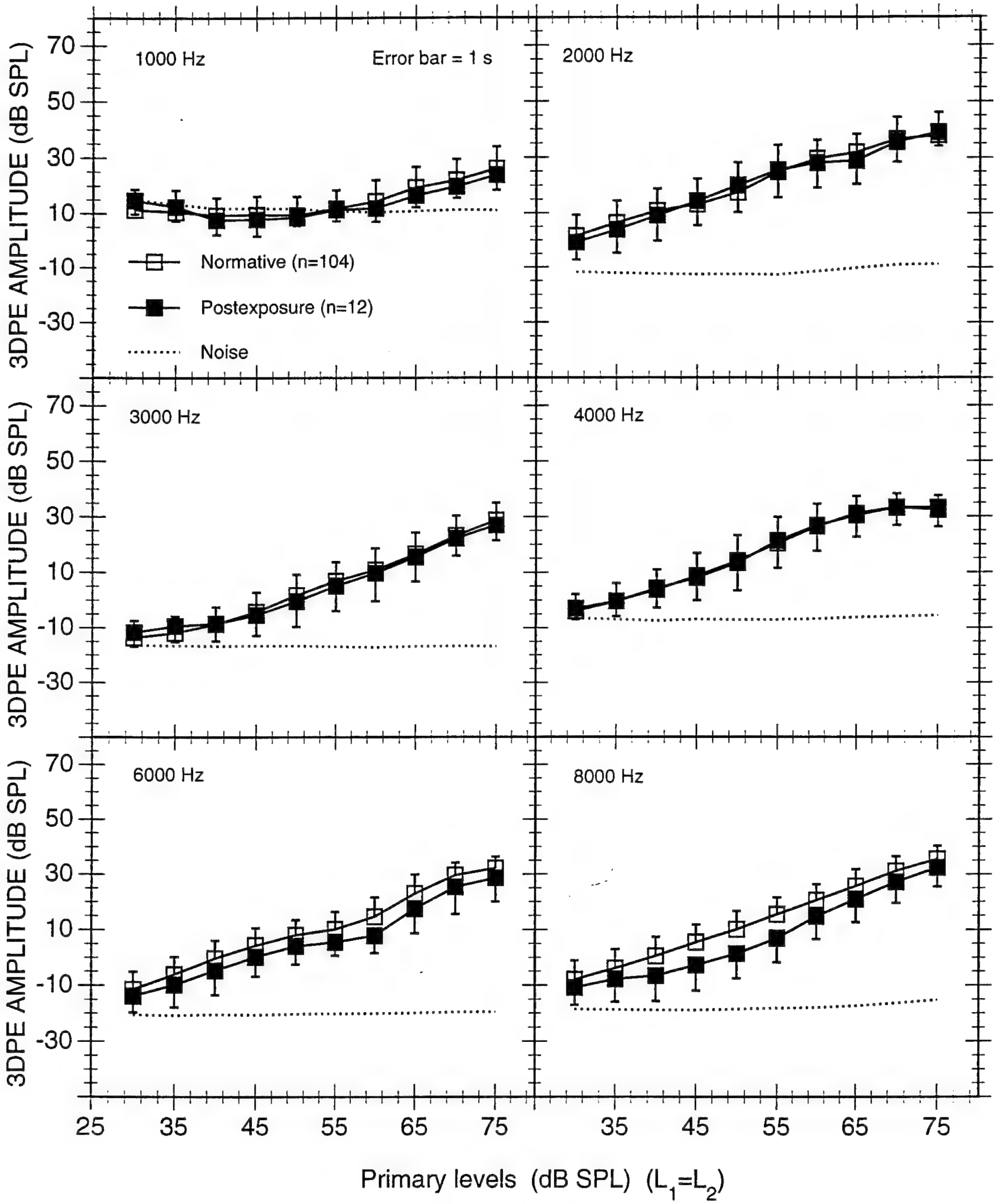

Figure 27. The mean postexposure (150 dB peak SPL, 100X, Source I) input/output functions at the indicated test frequencies ($f = \sqrt{f_1 f_2}$) compared with the mean normative functions. Error bars represent one standard deviation.

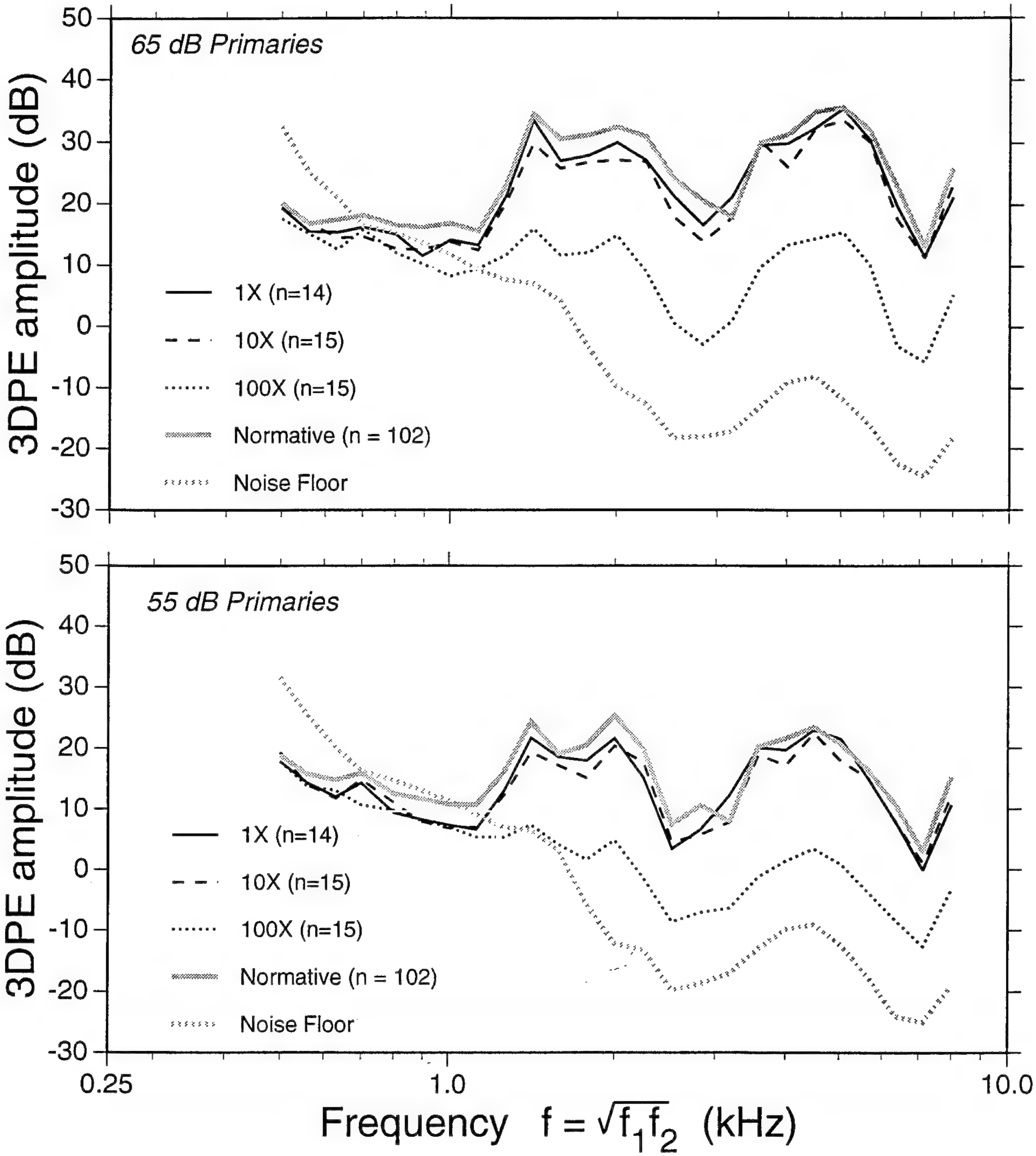

Figure 28. The group mean DPEgrams from three groups of chinchillas recorded at least 30 days after being exposed to 1, 10, or 100 reverberant blast waves from Source I at 155 dB peak SPL. The solid gray line represents the DPEgram from a normative group of 102 monaural chinchillas. The dotted gray line represents the noise floor from the normative sample.

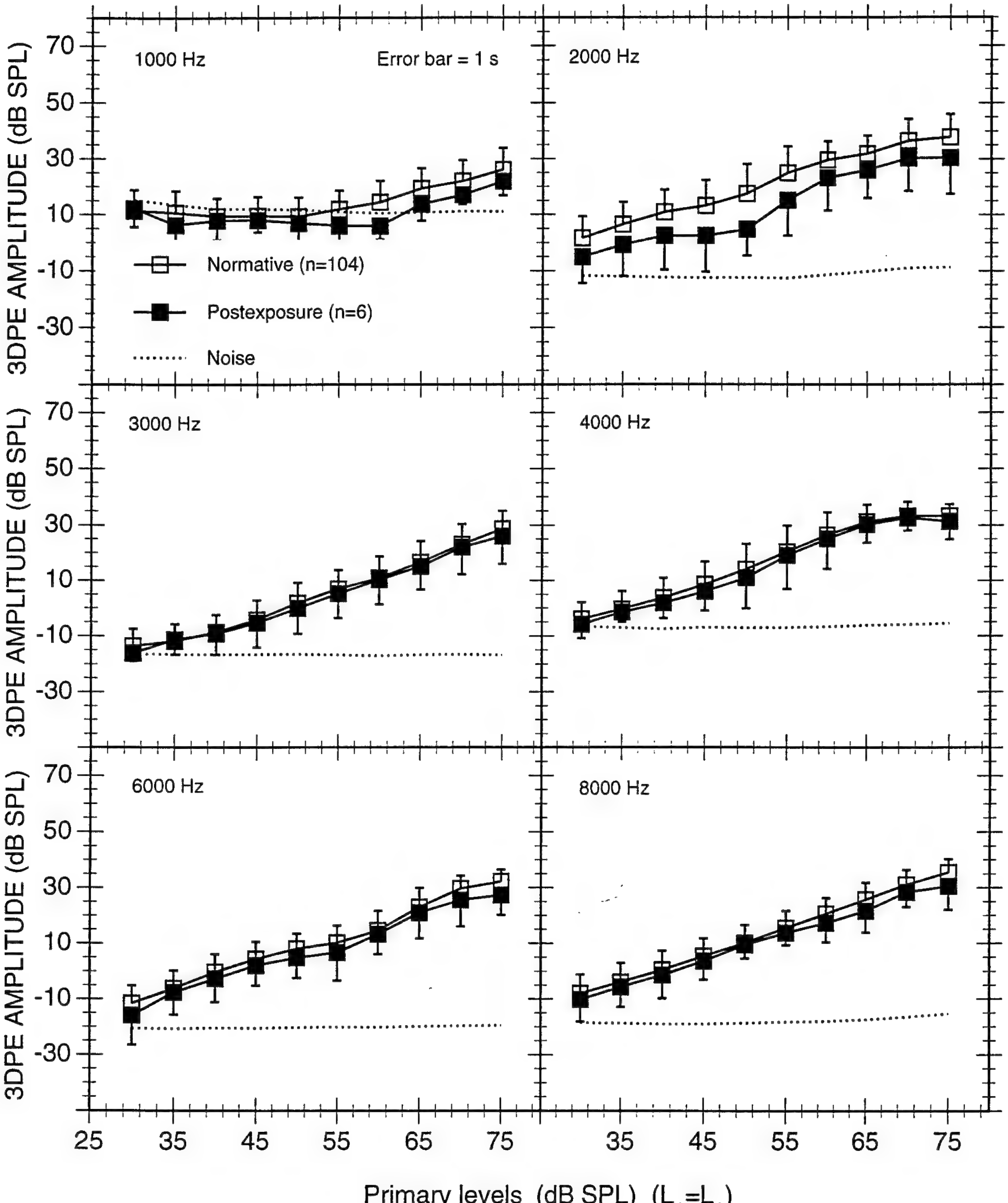

Figure 29. The mean postexposure (155 dB peak SPL, 1X, Source I) input/output functions at the indicated test frequencies ($f = \sqrt{f_1 f_2}$) compared with the mean normative functions. Error bars represent one standard deviation.

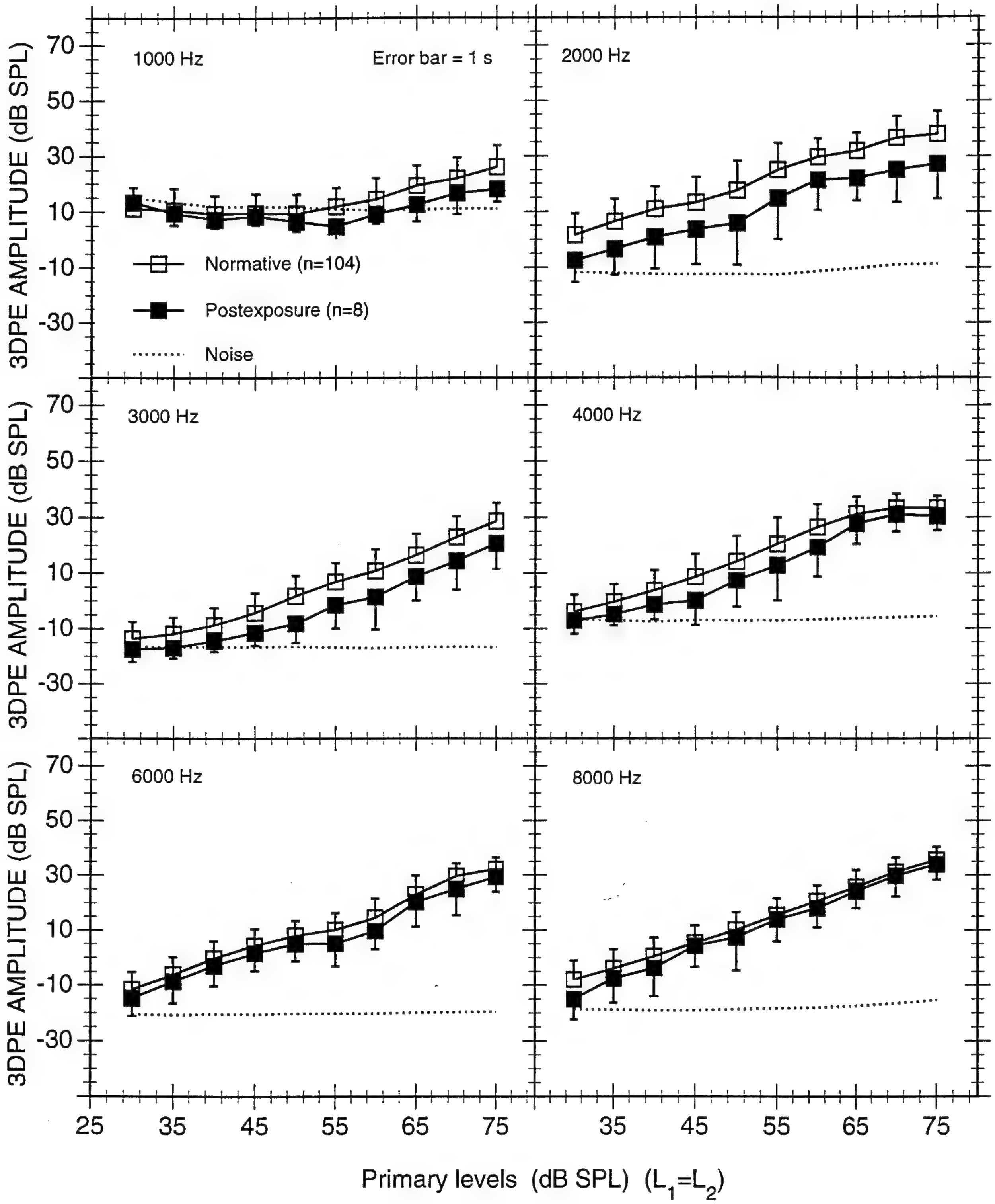

Figure 30. The mean postexposure (155 dB peak SPL, 10X, Source I) input/output functions at the indicated test frequencies ($f = \sqrt{f_1 f_2}$) compared with the mean normative functions. Error bars represent one standard deviation.

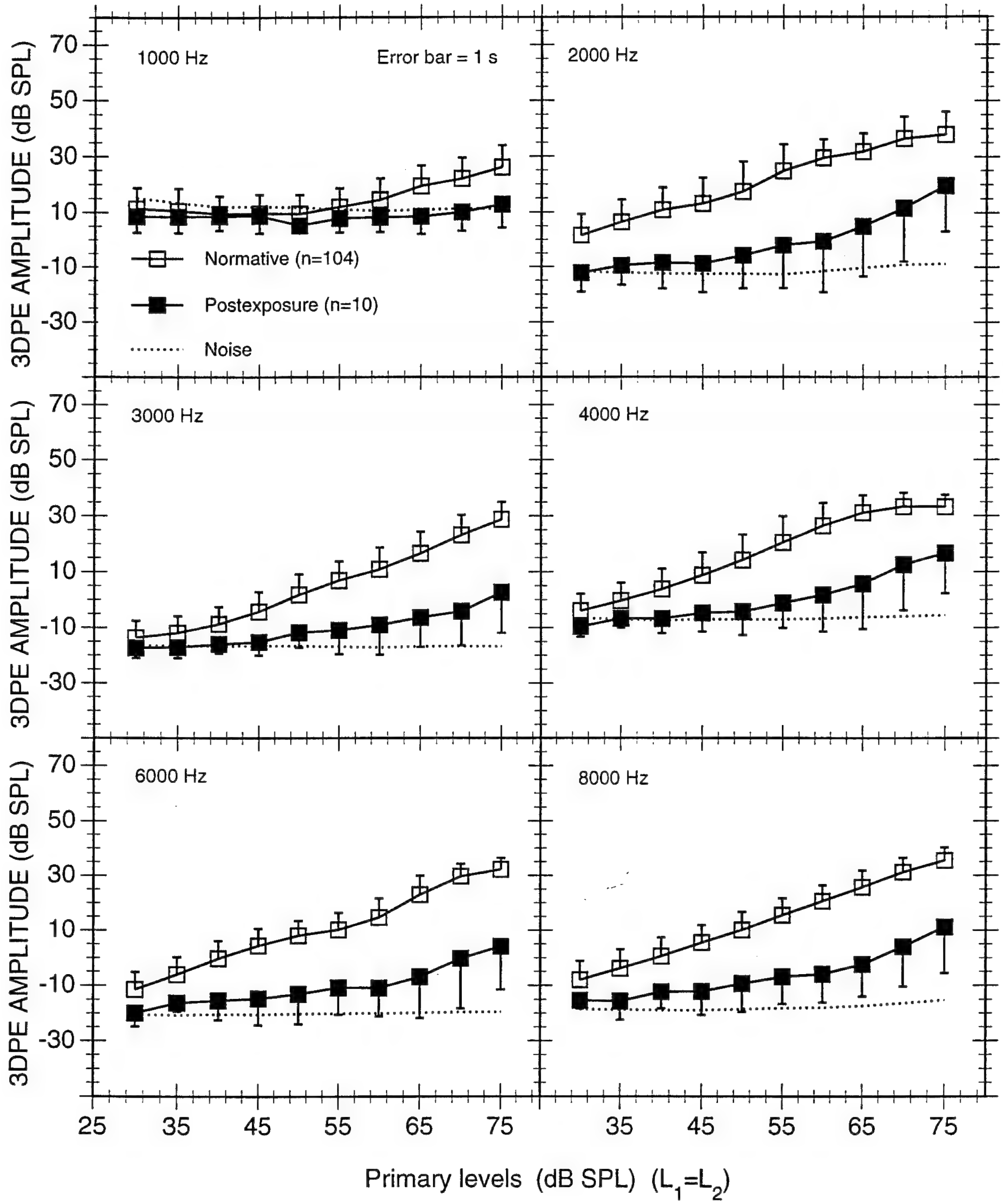

Figure 31. The mean postexposure (155 dB peak SPL, 100X, Source I) input/output functions at the indicated test frequencies ($f = \sqrt{f_1 f_2}$) compared with the mean normative functions. Error bars represent one standard deviation.

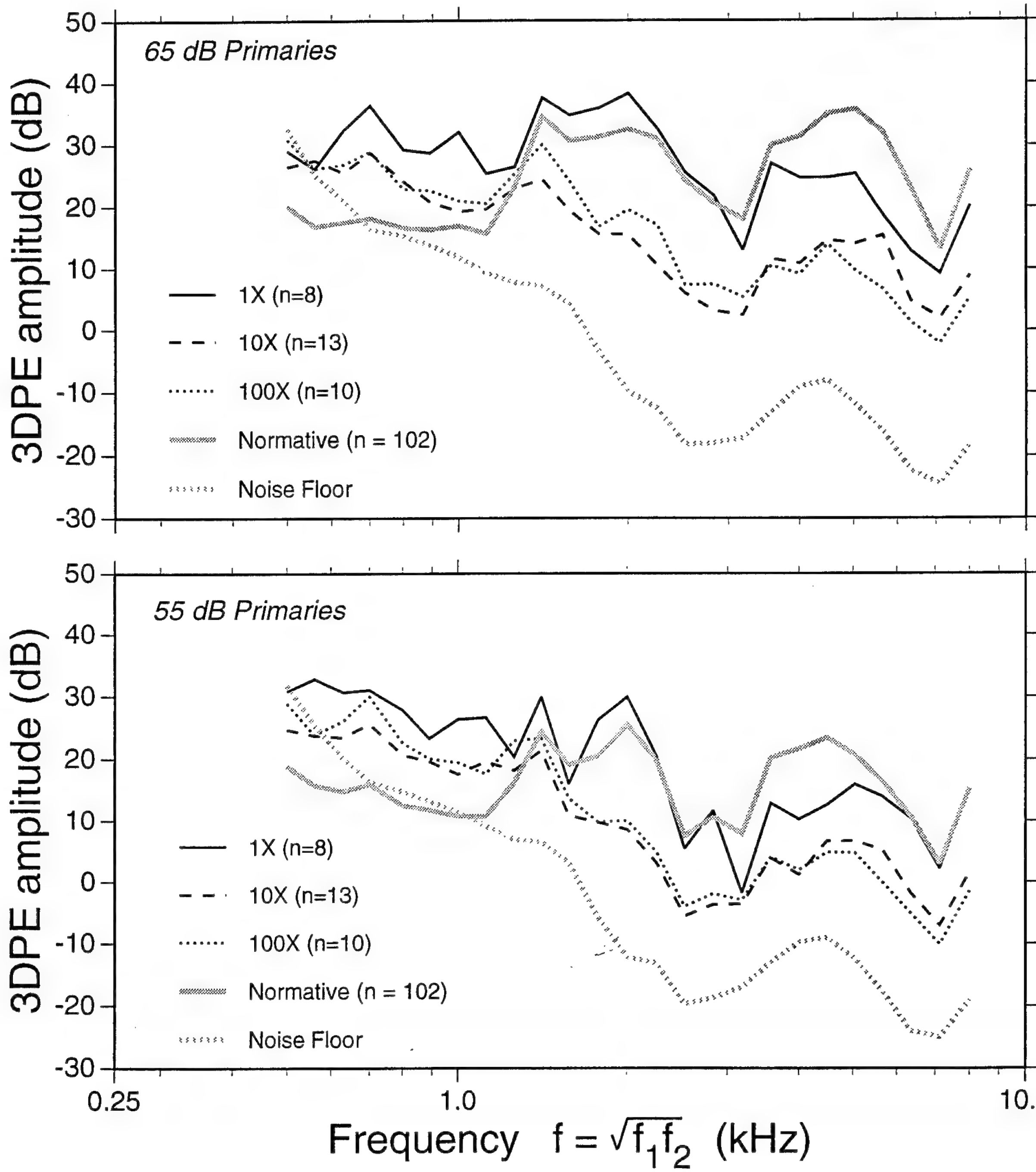

Figure 32. The group mean DPEgrams from three groups of chinchillas recorded at least 30 days after being exposed to 1, 10, or 100 reverberant blast waves from Source I at 160 dB peak SPL. The solid gray line represents the DPEgram from a normative group of 102 monaural chinchillas. The dotted gray line represents the noise floor from the normative sample.

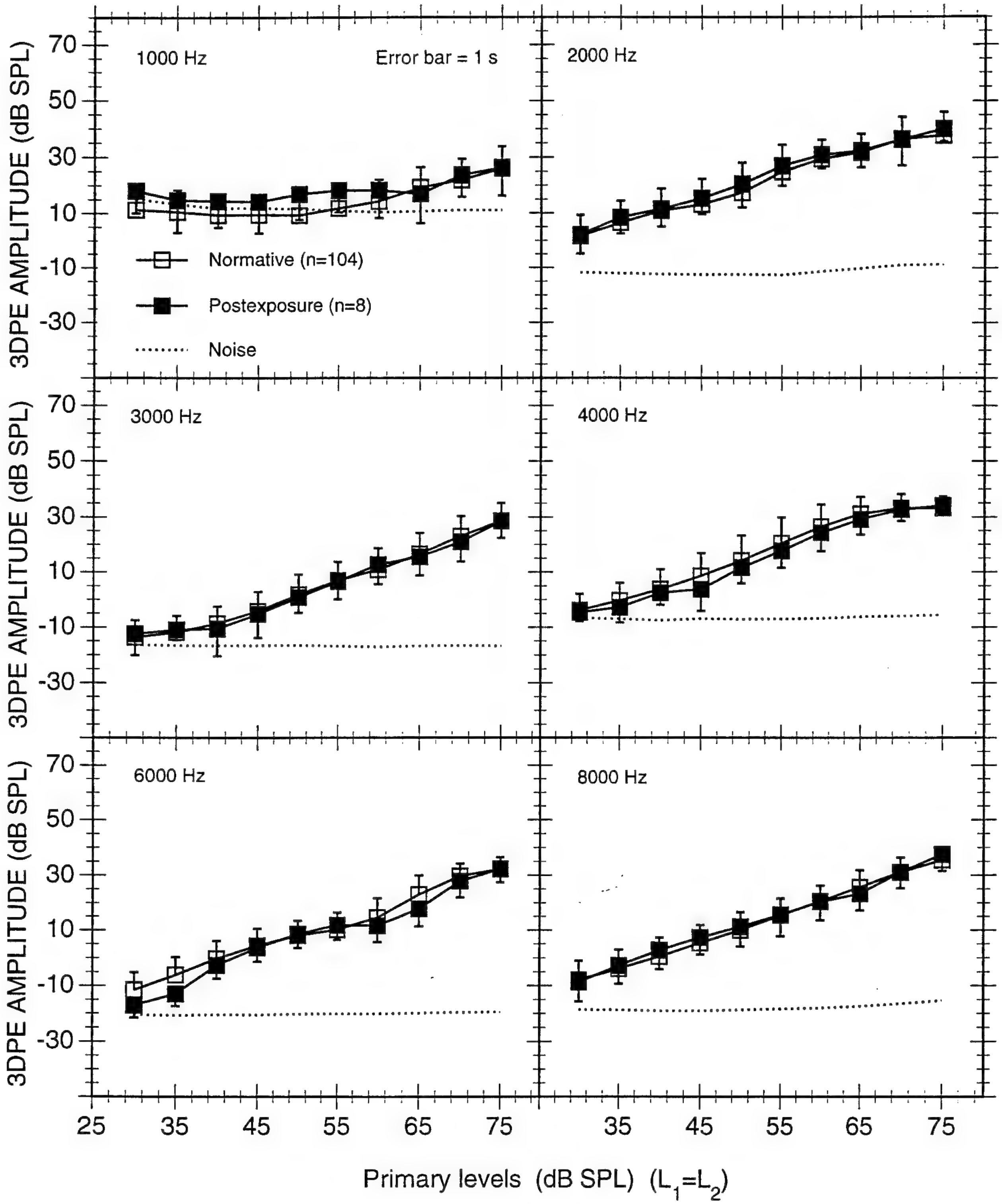

Figure 33. The mean postexposure (160 dB peak SPL, 1X, Source I) input/output functions at the indicated test frequencies ($f = \sqrt{f_1 f_2}$) compared with the mean normative functions. Error bars represent one standard deviation.

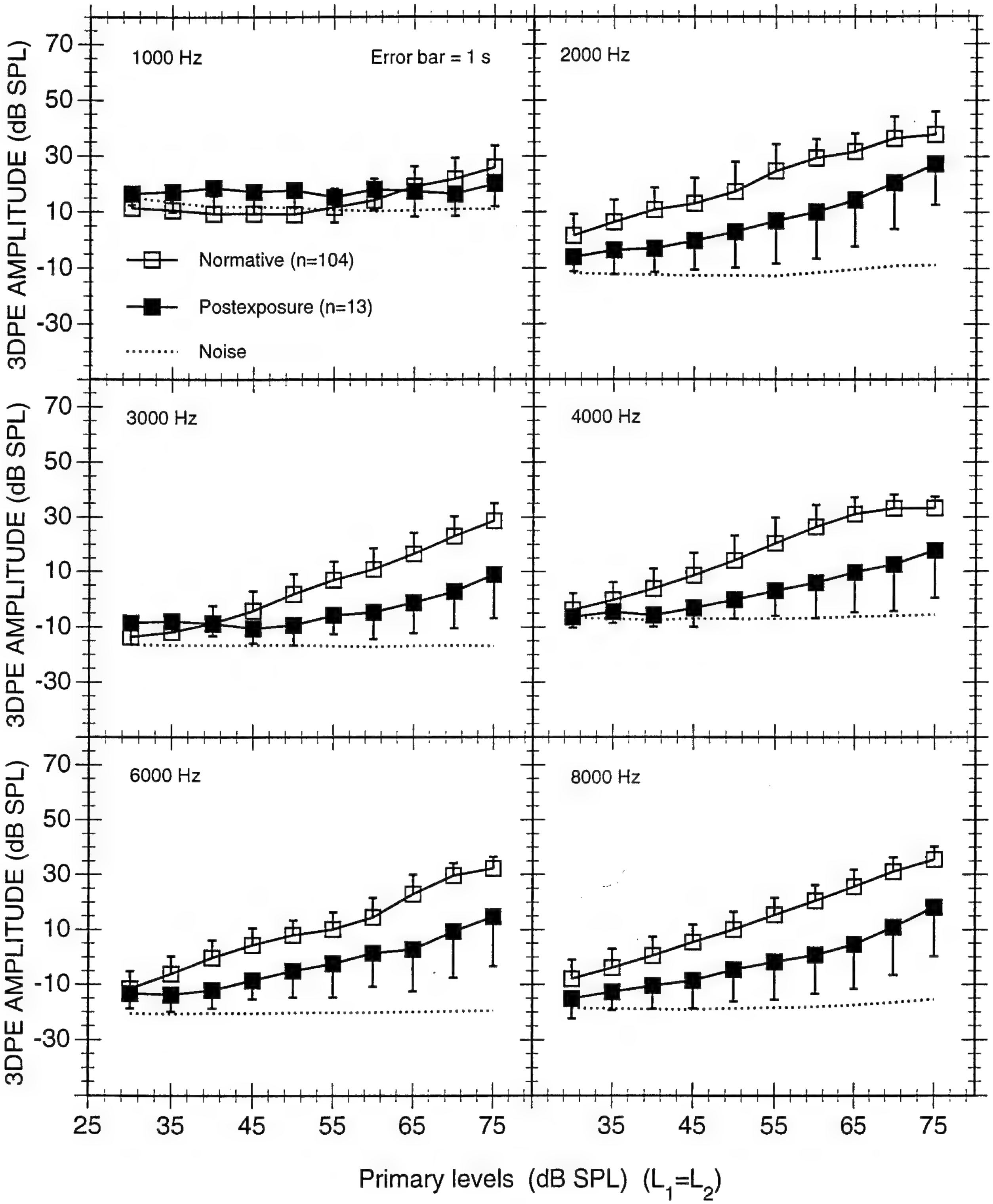

Figure 34. The mean postexposure (160 dB peak SPL, 10X, Source I) input/output functions at the indicated test frequencies ($f = \sqrt{f_1 f_2}$) compared with the mean normative functions. Error bars represent one standard deviation.

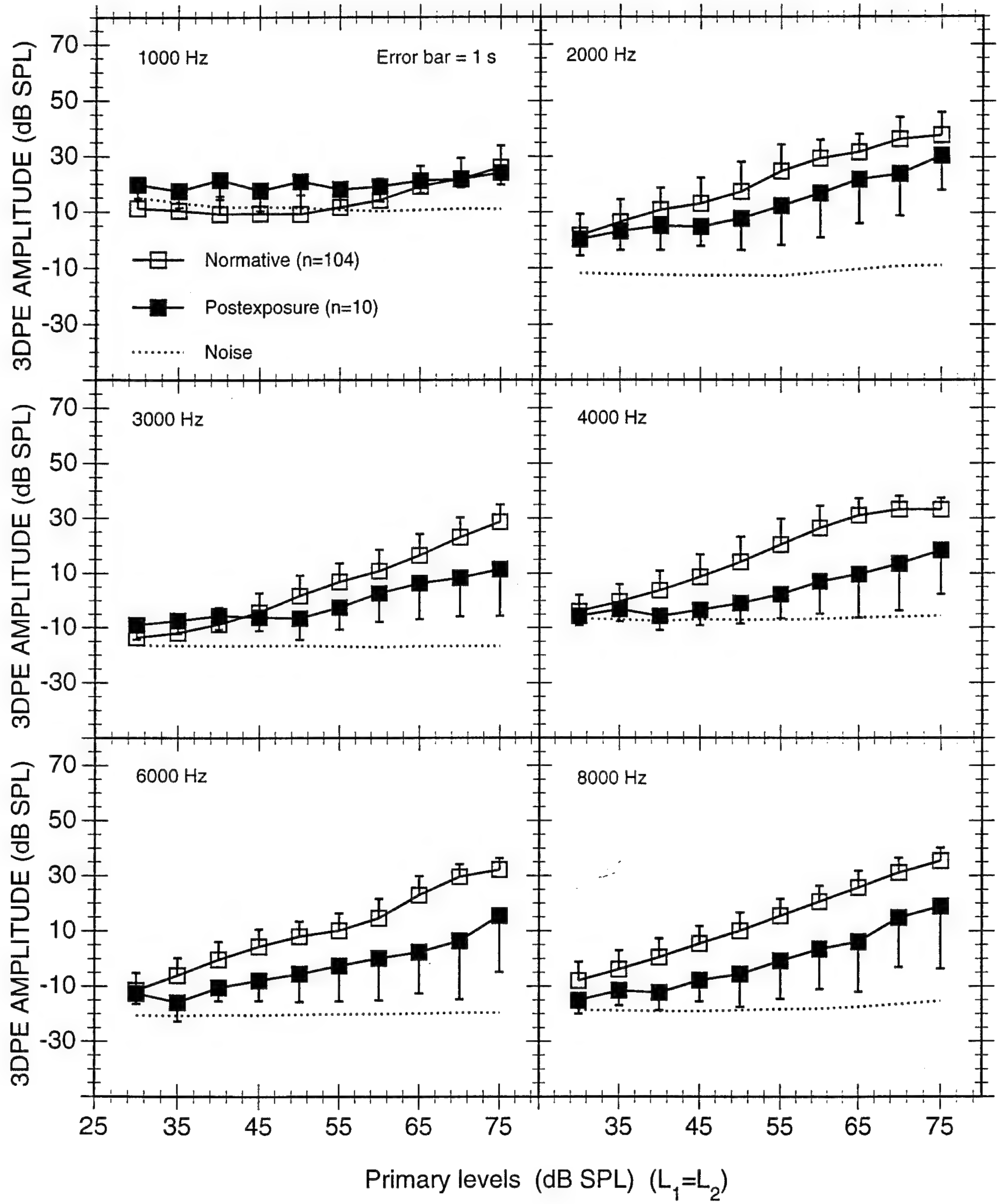

Figure 35. The mean postexposure (160 dB peak SPL, 100X, Source I) input/output functions at the indicated test frequencies ($f = \sqrt{f_1 f_2}$) compared with the mean normative functions. Error bars represent one standard deviation.

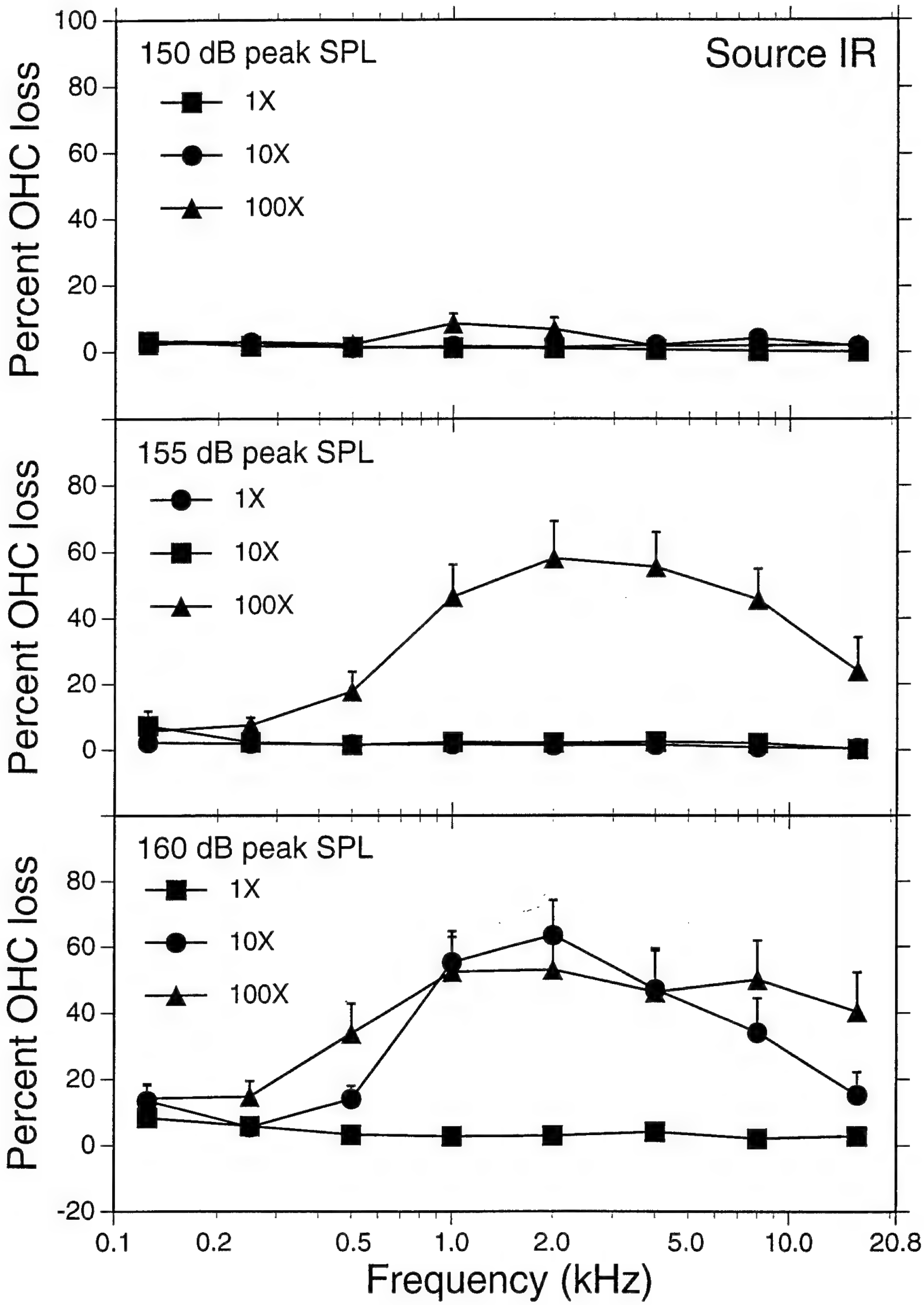

Figure 36. The group mean percent outer hair cell (OHC) loss measured 30 days after exposure to reverberant blast waves produced by Source I at each of the nine conditions indicated. Error bars represent one standard error of the mean.

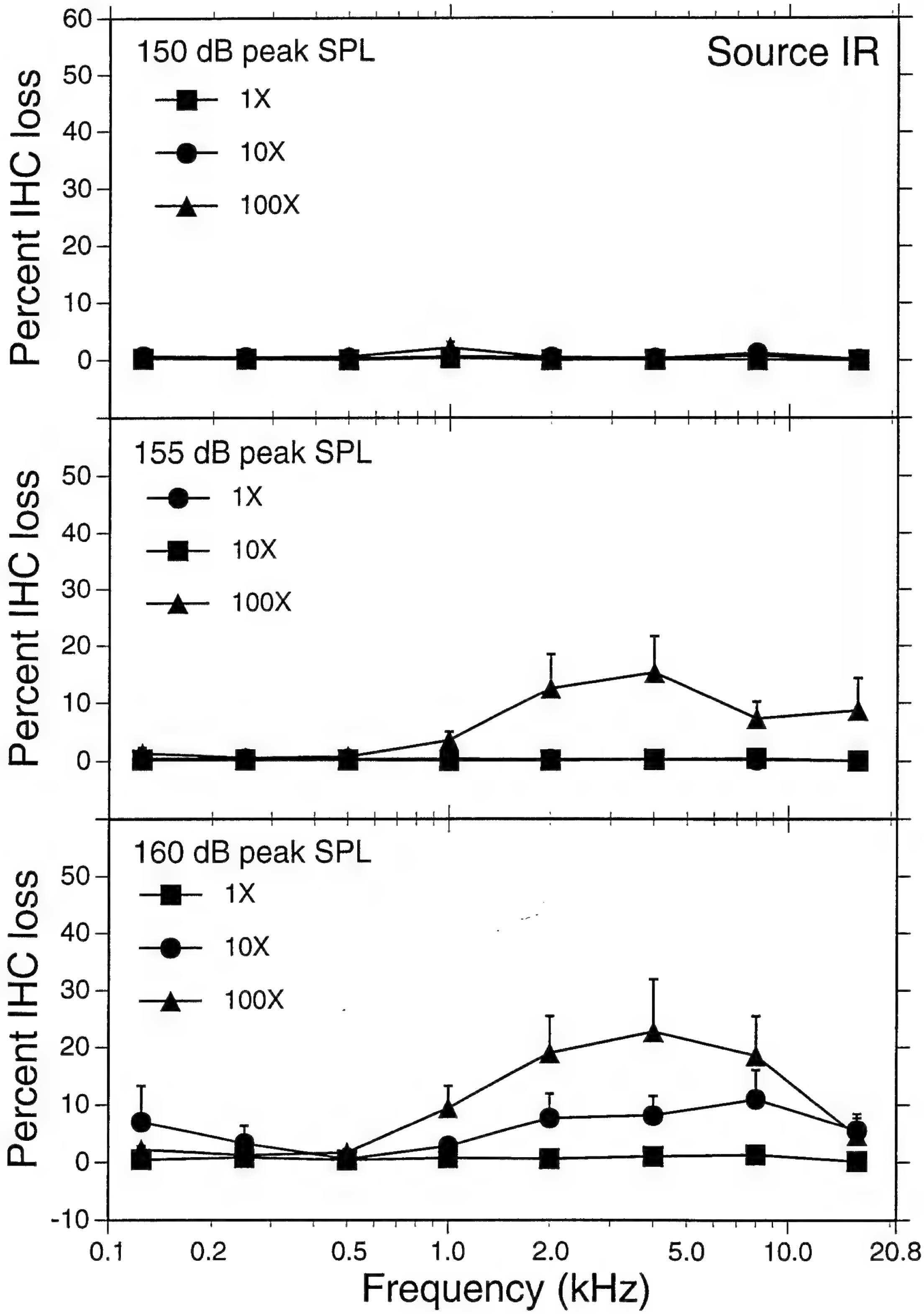

Figure 37. The group mean percent inner hair cell (IHC) loss measured 30 days after exposure to reverberant blast waves produced by Source I at each of the nine conditions indicated. Error bars represent one standard error of the mean.

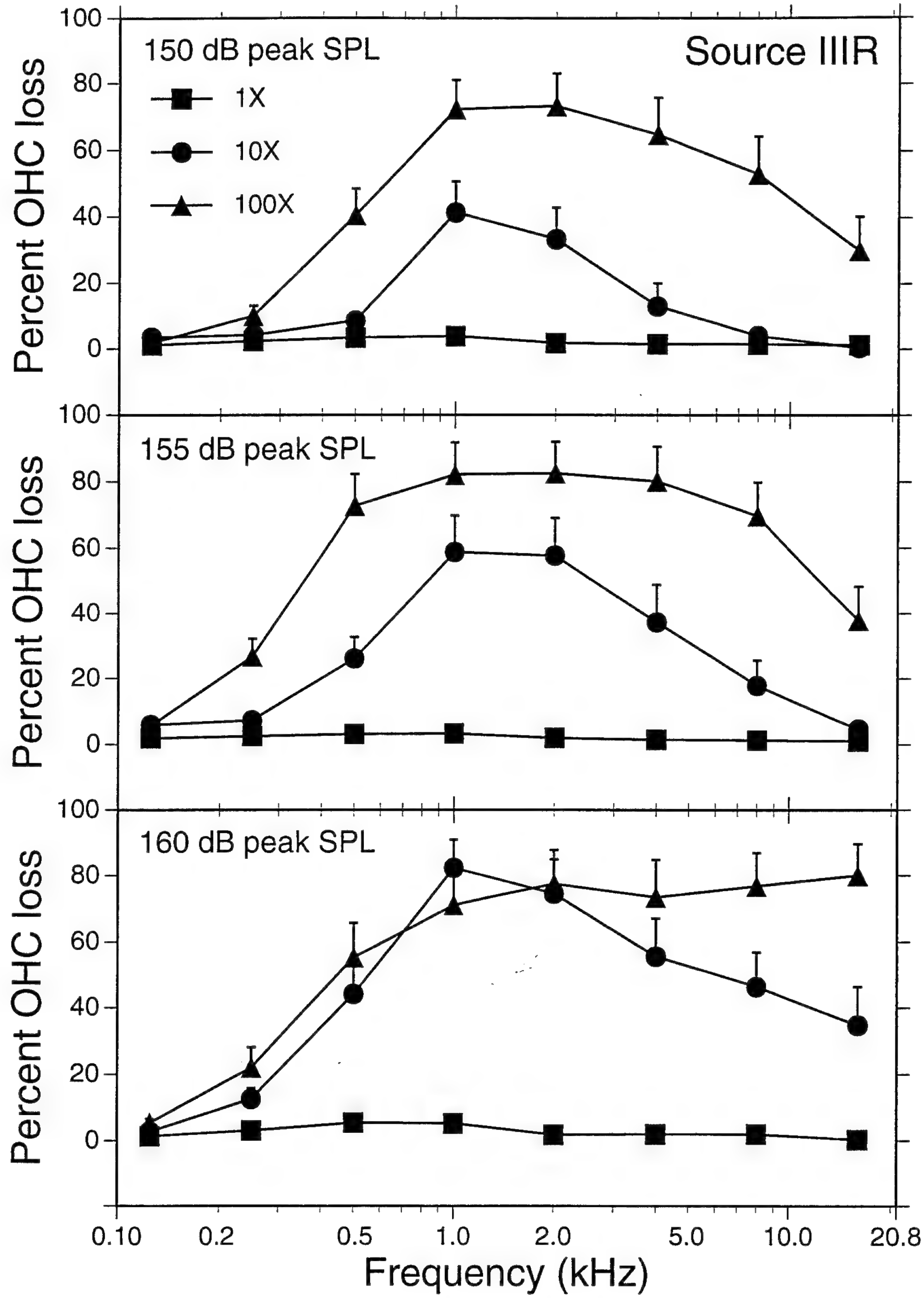

Figure 38. The group mean percent outer hair cell (OHC) loss measured 30 days after exposure to reverberant blast waves produced by Source III at each of the nine conditions indicated. Error bars represent one standard error of the mean.

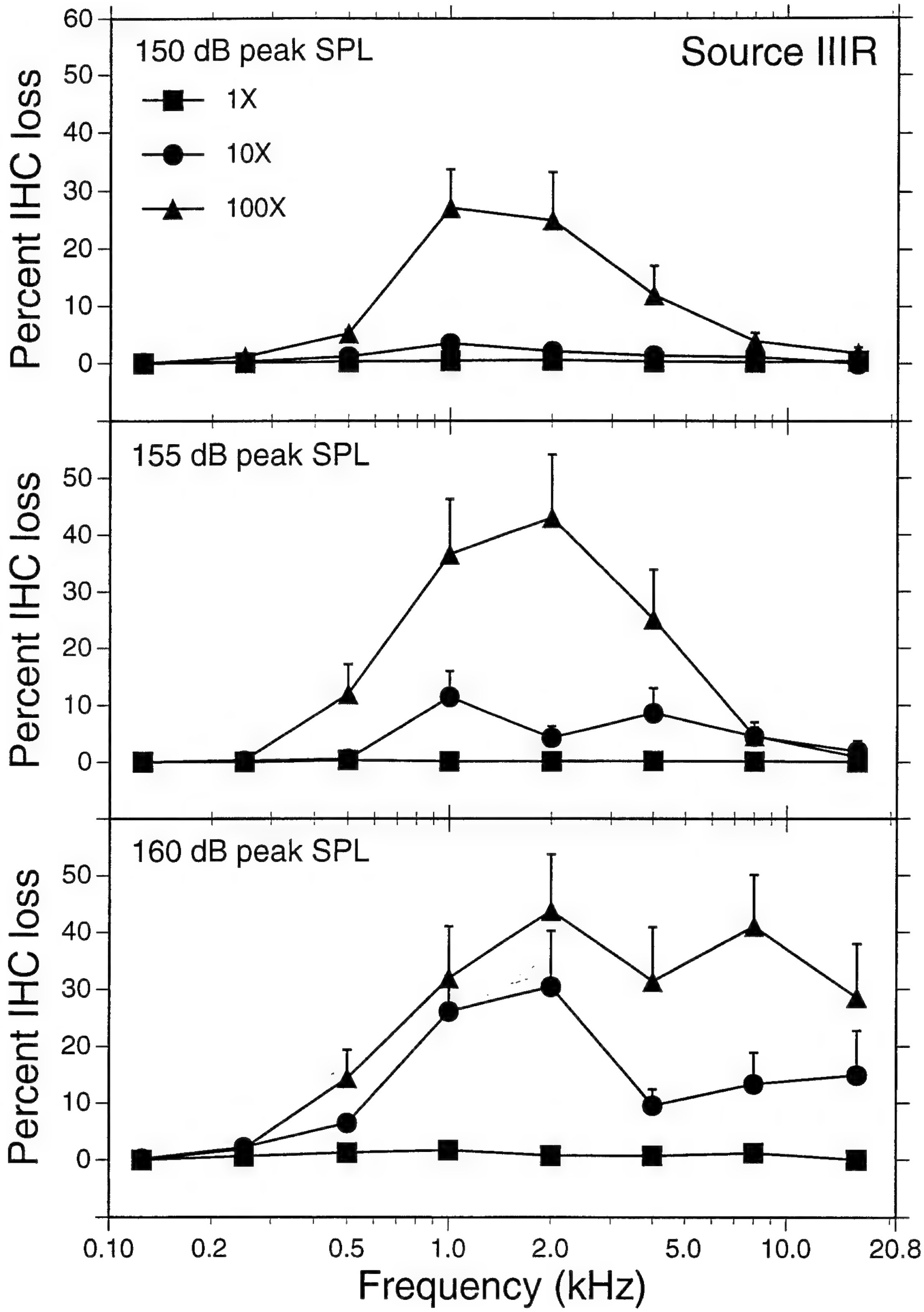

Figure 39. The group mean percent inner hair cell (IHC) loss measured 30 days after exposure to reverberant blast waves produced by Source III at each of the nine conditions indicated. Error bars represent one standard error of the mean.

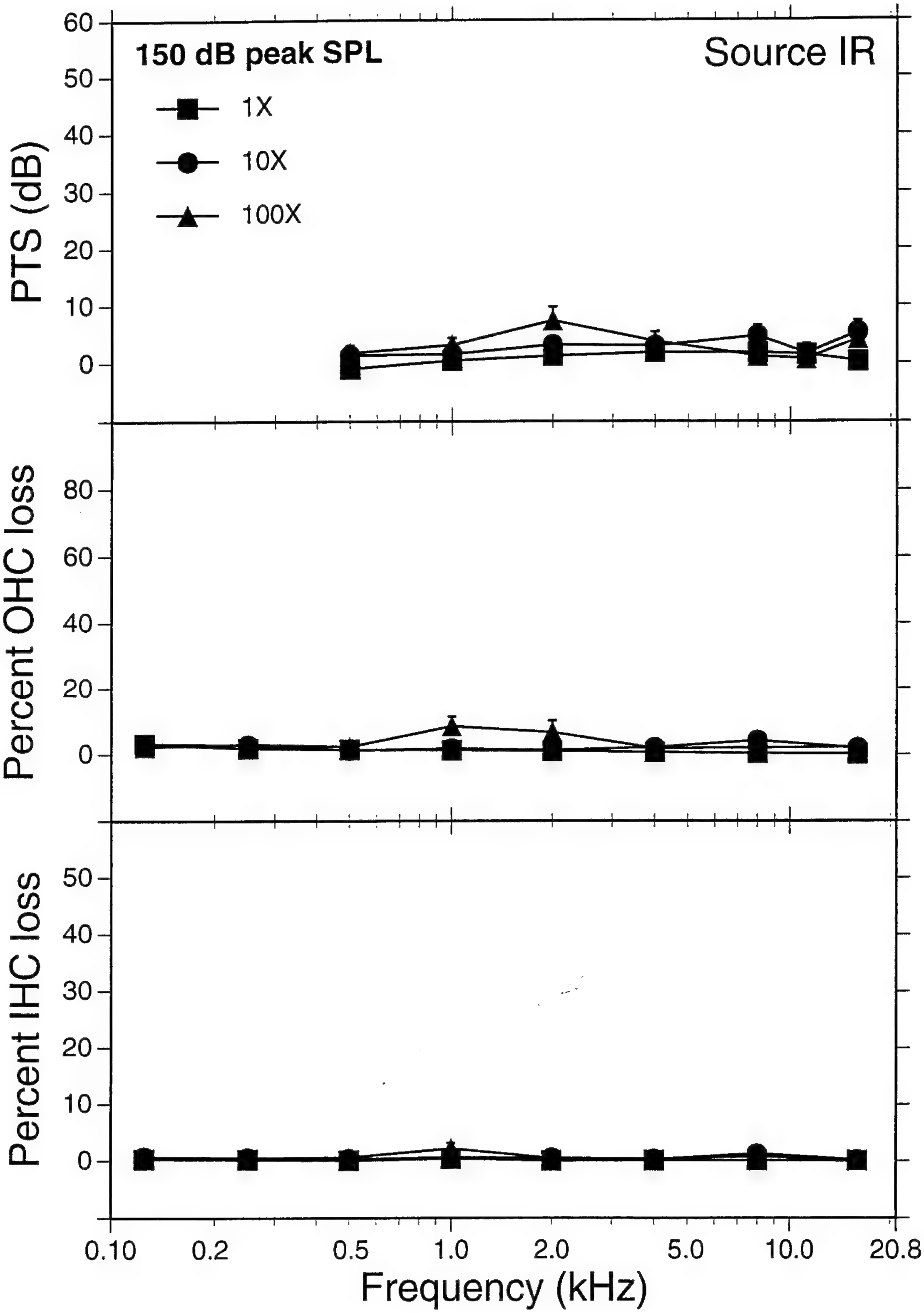

Figure 40. The group mean permanent threshold shift (PTS, top panel) audiograms, percent outer hair cell loss (OHC, center panel), and percent inner hair cell loss (IHC, bottom panel) measured 30 days after exposure to 150 dB peak SPL reverberant blast waves produced by Source I at each of the three conditions indicated. Error bars represent one standard error of the mean.

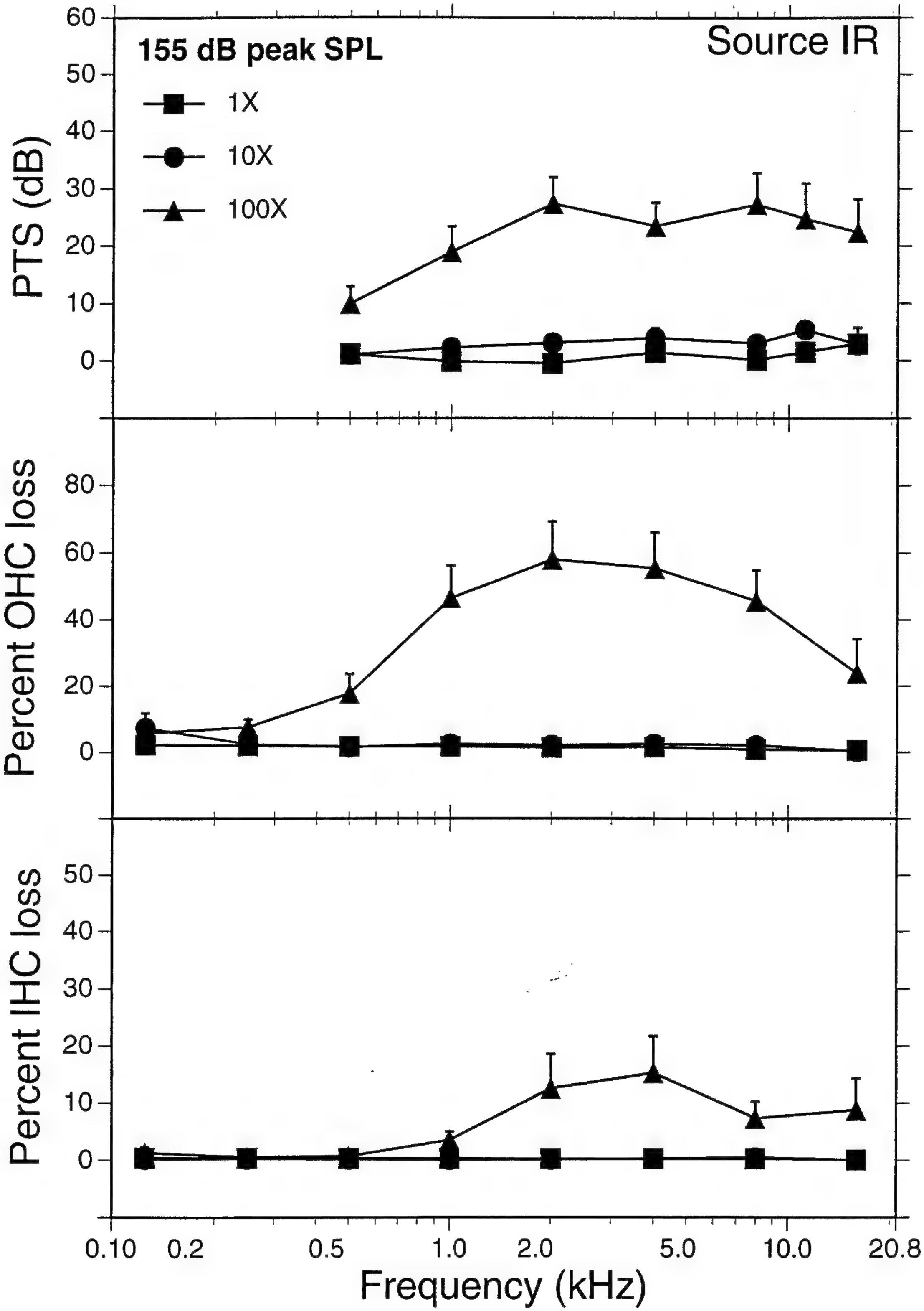

Figure 41. The group mean permanent threshold shift (PTS, top panel) audiograms, percent outer hair cell loss (OHC, center panel), and percent inner hair cell loss (IHC, bottom panel) measured 30 days after exposure to 155 dB peak SPL reverberant blast waves produced by Source I at each of the three conditions indicated. Error bars represent one standard error of the mean.

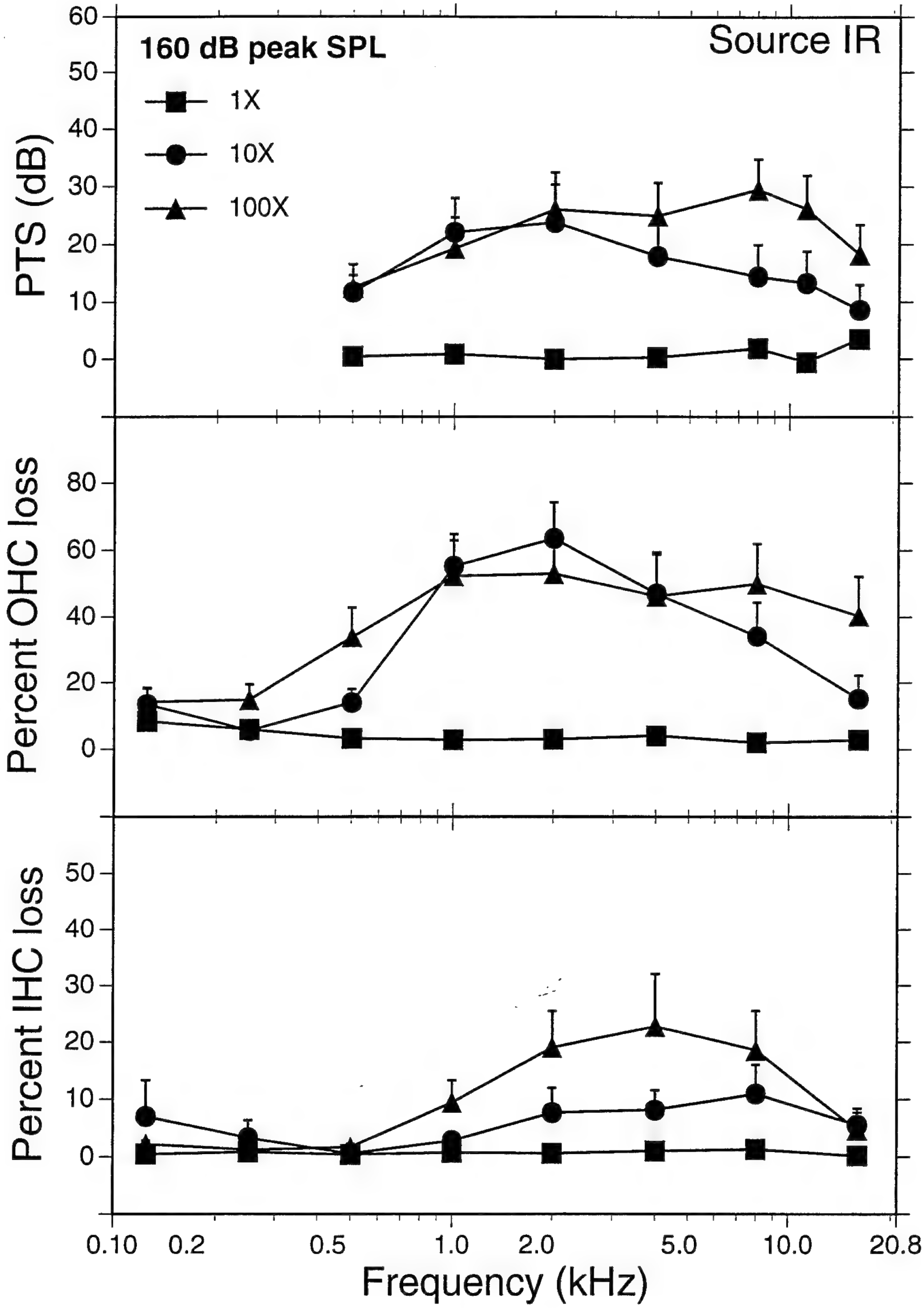

Figure 42. The group mean permanent threshold shift (PTS, top panel) audiograms, percent outer hair cell loss (OHC, center panel), and percent inner hair cell loss (IHC, bottom panel) measured 30 days after exposure to 160 dB peak SPL reverberant blast waves produced by Source I at each of the three conditions indicated. Error bars represent one standard error of the mean.

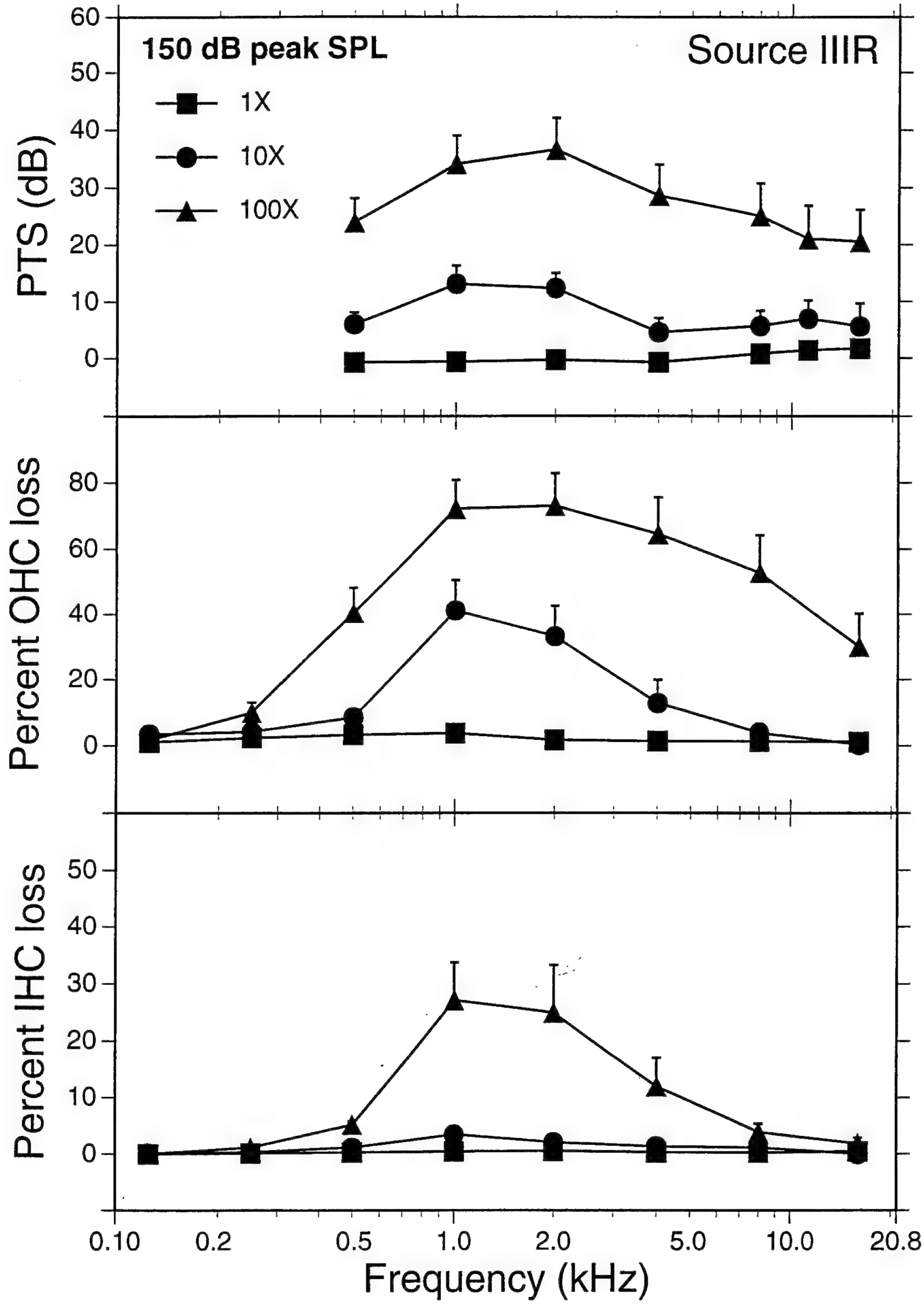

Figure 43. The group mean permanent threshold shift (PTS, top panel) audiograms, percent outer hair cell loss (OHC, center panel), and percent inner hair cell loss (IHC, bottom panel) measured 30 days after exposure to 150 dB peak SPL reverberant blast waves produced by Source III at each of the three conditions indicated. Error bars represent one standard error of the mean.

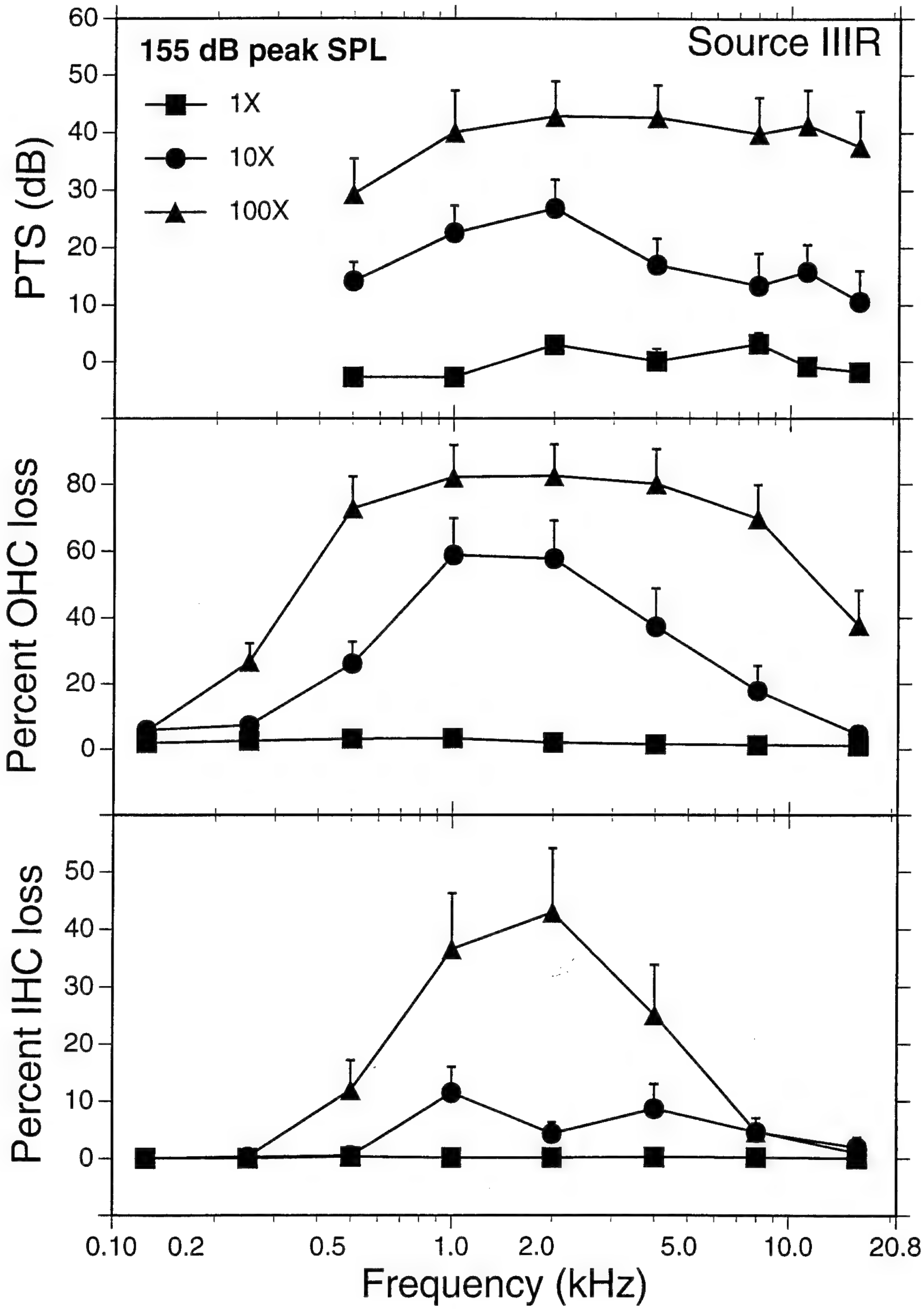

Figure 44. The group mean permanent threshold shift (PTS, top panel) audiograms, percent outer hair cell loss (OHC, center panel), and percent inner hair cell loss (IHC, bottom panel) measured 30 days after exposure to 155 dB peak SPL reverberant blast waves produced by Source III at each of the three conditions indicated. Error bars represent one standard error of the mean.

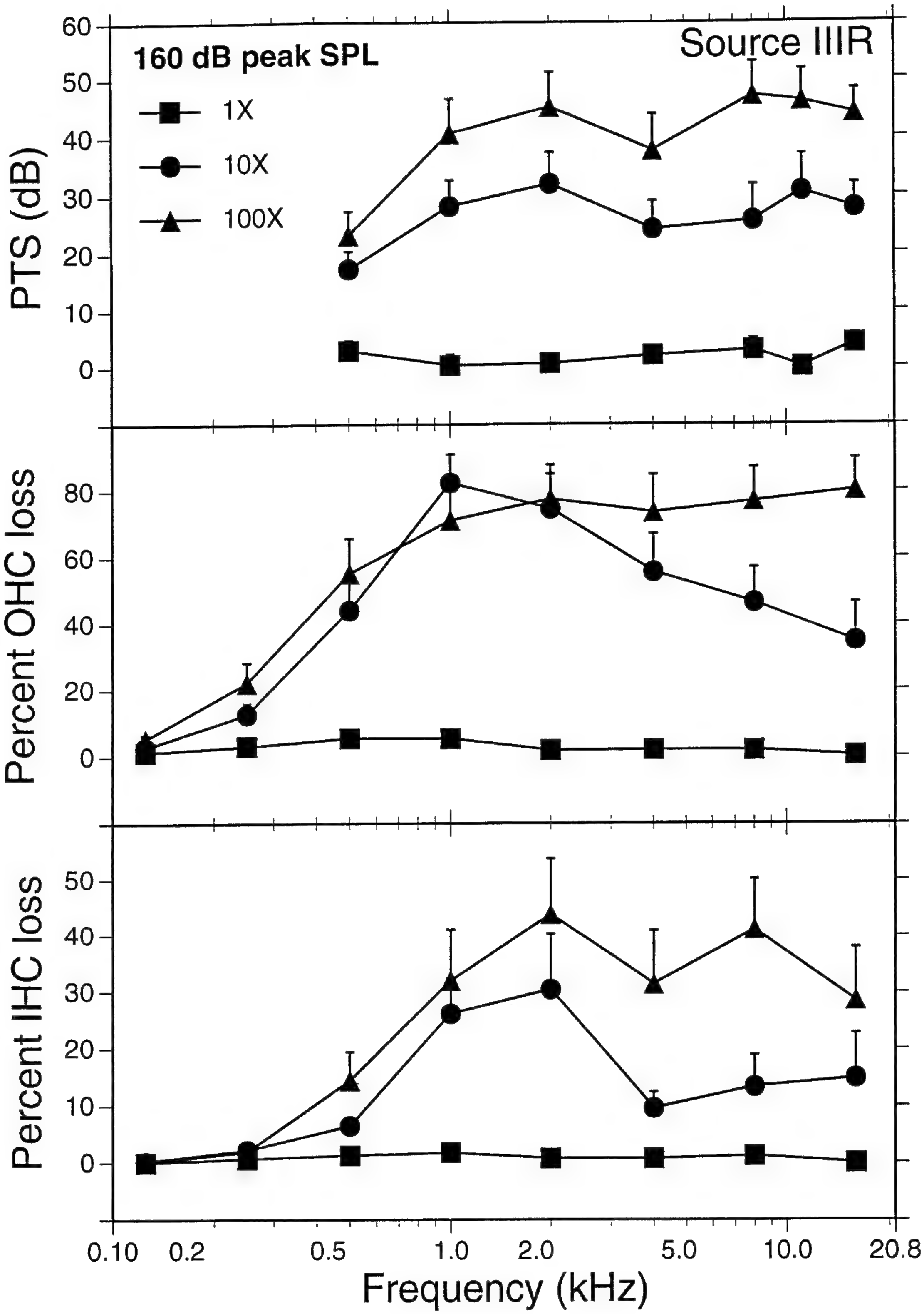

Figure 45. The group mean permanent threshold shift (PTS, top panel) audiograms, percent outer hair cell loss (OHC, center panel), and percent inner hair cell loss (IHC, bottom panel) measured 30 days after exposure to 160 dB peak SPL reverberant blast waves produced by Source III at each of the three conditions indicated. Error bars represent one standard error of the mean.

Figure 46 and 47 show the group mean PTS measured at 1.0, 2.0, and 4.0 kHz ($\overline{PTS}_{1,2,4}$) and the total group mean OHC loss respectively plotted as a function of the total unweighted, A-weighted and P-weighted SEL. A linear regression analysis shows better correlations of $\overline{PTS}_{1,2,4}$, and total OHC loss with the weighted SEL than with the unweighted SEL. The only difference between A- and P-weighting appears to be approximately 3 dB higher SEL threshold for the onset of trauma using the P weighting function.

Figures 48 through 53 summarize the 3DPE data. The change (Δ) in the 3DPE output after exposure for 55 and 65 dB primaries is compared with the mean PTS for those same animals in Figures 48-50. The same 3DPE data is compared with the mean OHC losses of these animals in Figures 51-53. For the 150 dB peak SPL exposures there was little PTS or OHC loss and no significant changes in 3DPEs. As the exposures began to cause changes in hearing function and sensory cell lesions, the 3DPEs showed changes that paralleled the PTS and OHC losses above 1.0 kHz. At and below 1.0 kHz, the 3DPEs could not be extracted from the noise floor with the instrument in use. Thus, based upon these preliminary 3DPE data it appears that distortion product emissions may be of value in diagnosing noise-induced hearing loss and cochlear pathology.

V. RECOMMENDATIONS FOR FURTHER DATA ANALYSIS

An understanding of how the various parameters of an impulse noise (blast wave) exposure affect hearing is critical to our abilities to evaluate noise exposures for the purposes of hearing conservation practice and to the design of hearing protective devices. Over the past approximately 15 years, professional staff of the Auditory Research Laboratories (ARL) of the State University of New York at Plattsburgh and at the Callier Center at the University of Texas-Dallas have been engaged in DoD sponsored studies of the effects of blast wave (impulse noise) exposures on hearing. This work, which used the chinchilla as an animal model, was supported by grant and contracts USAMRDC DAMD17-80-C-0133, USAMRDC DAMD17-83-G-9555, USAMRDC DAMD17-86-C-6172 and USAMRDC DAMD17-91-C-1113. These experiments involved approximately 700 subjects exposed to 102 different blast wave exposures. These laboratories have also been involved in the design and analysis of the results of impulse noise experiments under contracts DAMD17-80-C-0109, DAMD17-86-C-6139 and DAMD17-91-C-1120 conducted at the USAARL facility. These experiments also used the chinchilla as the animal model and, to date, have involved 220 experimental subjects in 37 different exposure conditions.

Taken together, this research generated a very large amount of data (probably the largest such data base currently in existence) on how the various parameters of a blast wave exposure contribute to hearing loss. However, the ultimate value of this data base, involving nearly 1000 experimental animals, lies in the relations that can be developed between the exposure stimulus and the resultant trauma when the entire data base is integrated and analyzed as a single unit. In other words, it is important (a) to determine the interrelations among the various exposure parameters and the subsequent hearing loss and cochlear pathology and (b) use these interrelations to develop an approach to estimating the hazards posed by exposure to any blast wave pressure signatures. Such a comprehensive synthesis of the data will maximize the amount of information that can be obtained from the animal experiments; a goal in keeping with the principles of animal use

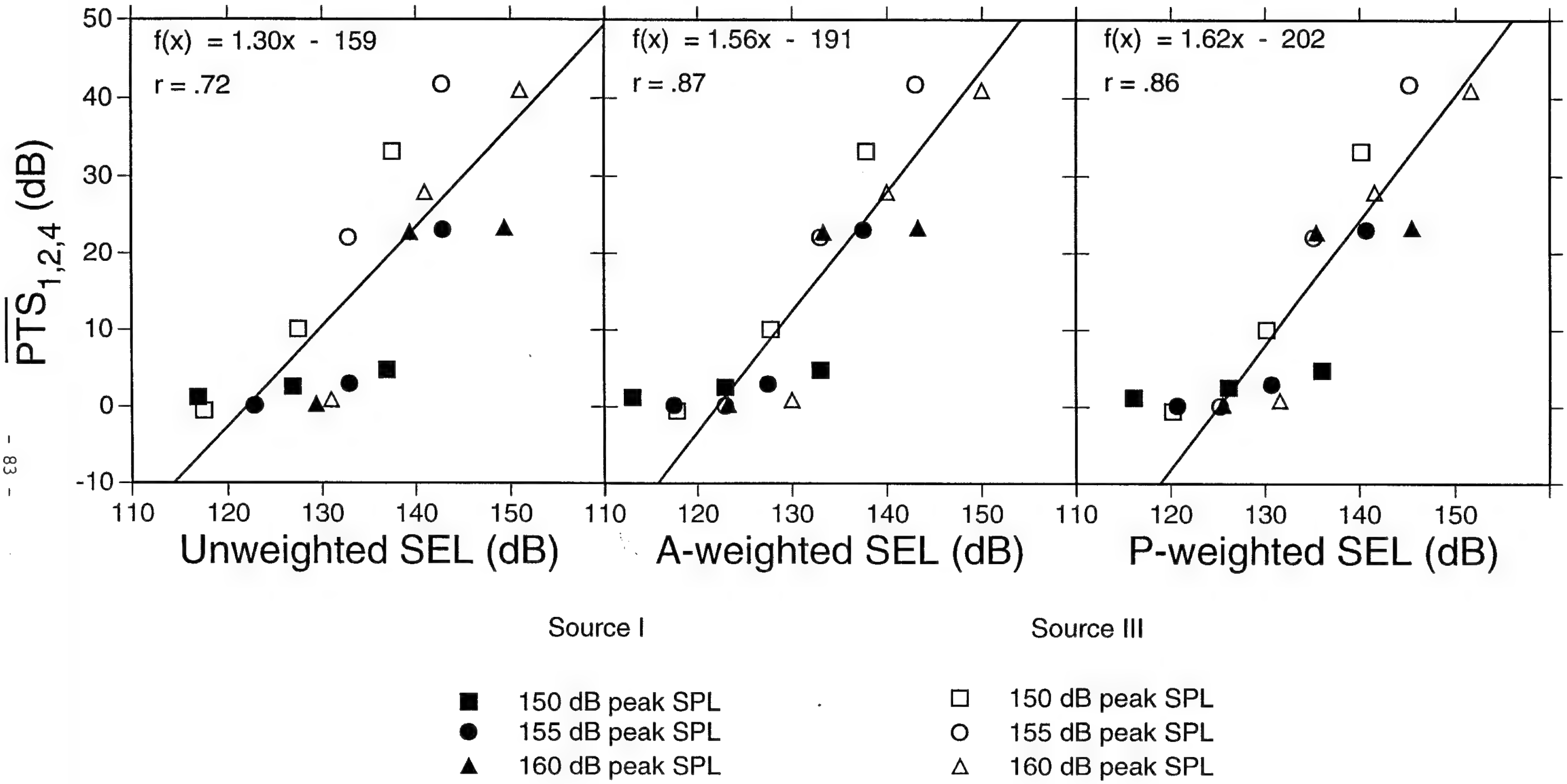

Figure 46. The group mean $PTS_{1,2,4}$ for each of the 18 exposure groups shown plotted against the unweighted SEL, the A-weighted SEL, and the P-weighted (Patterson et al., 1993) SEL.

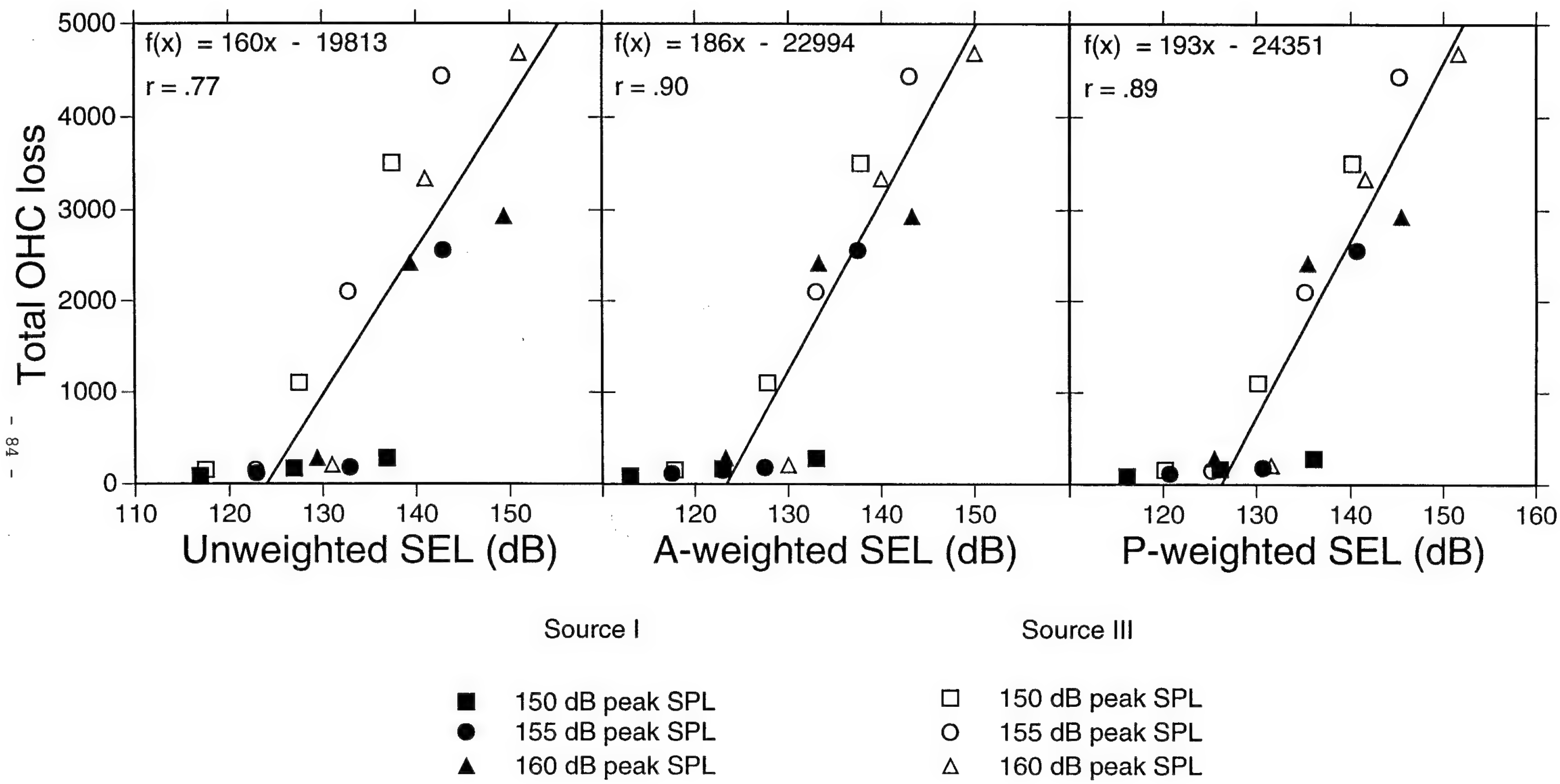

Figure 47. The group mean total outer hair cell (OHC) loss for each of the 18 exposure groups shown plotted against the unweighted SEL, the A-weighted SEL, and the P-weighted (Patterson et al., 1993) SEL.

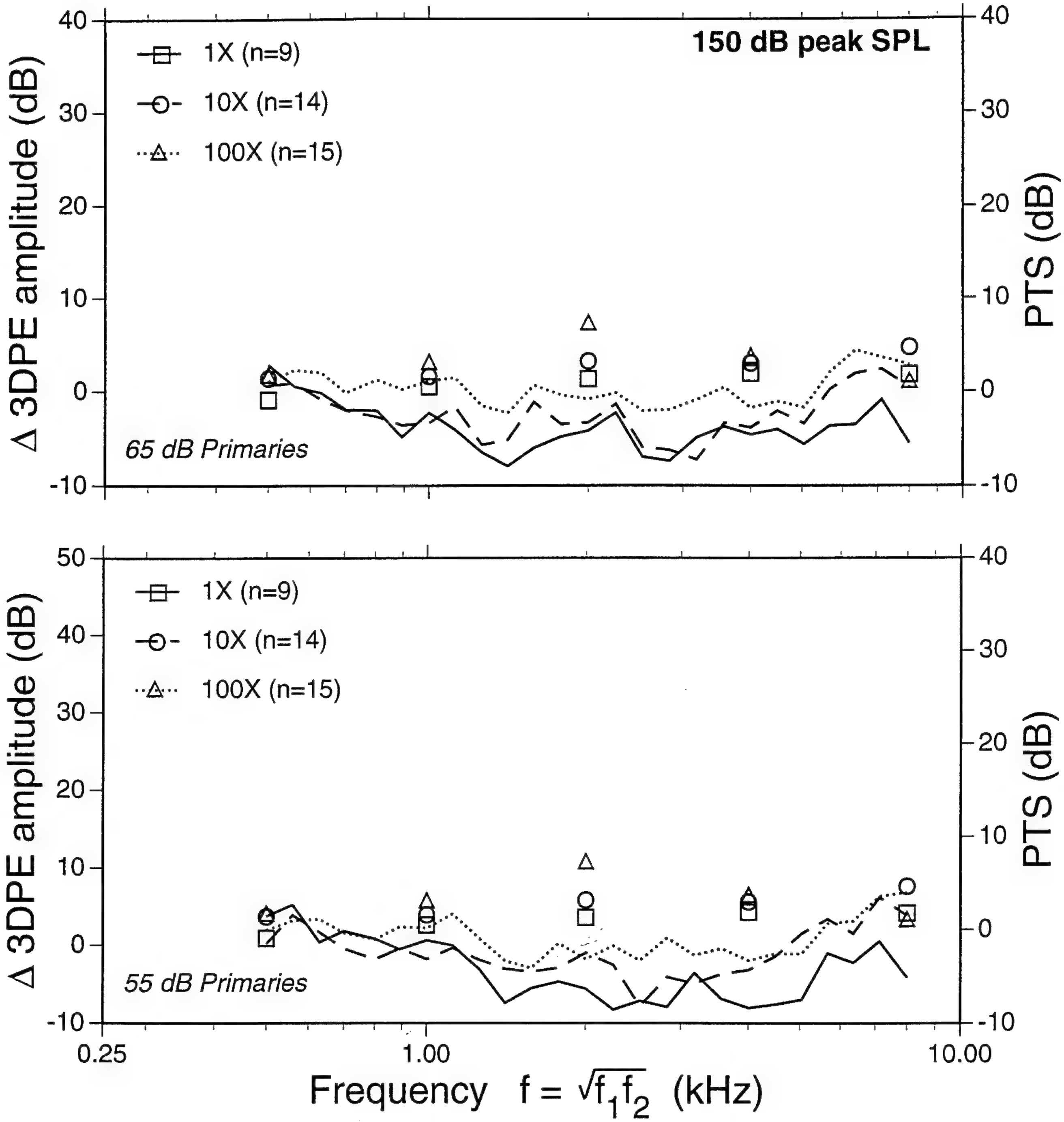

Figure 48. The group mean change in DPEgrams and average PTS from three groups of chinchillas at least 30 days after being exposed to 1, 10, or 100 reverberant blast waves from Source I at 150 dB peak SPL. Each line represents the difference between the normative (n=102) mean DPEgram and the mean experimental group DPEgram. The open symbols represent the mean PTS for each group at the specified frequencies.

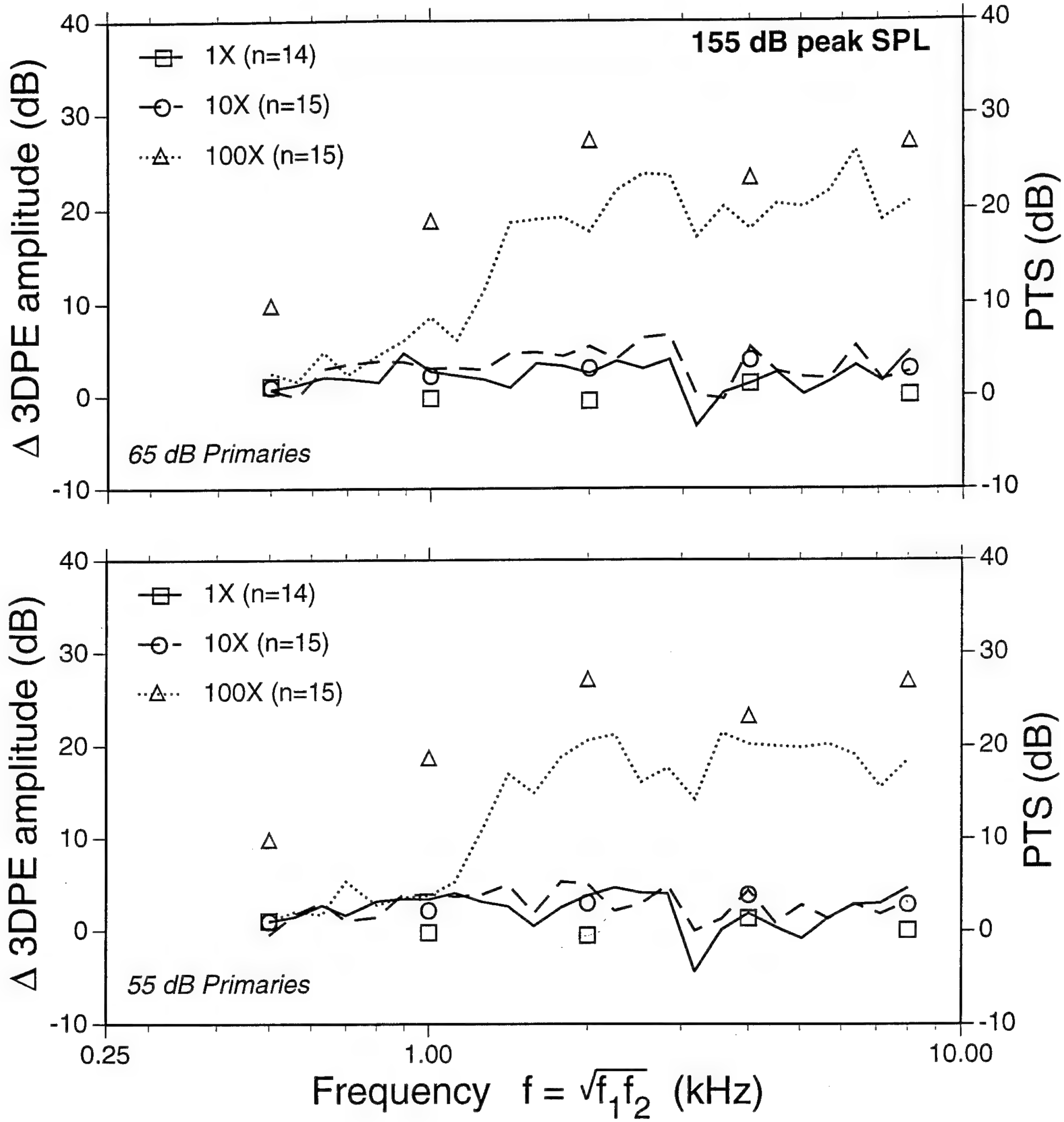

Figure 49. The group mean change in DPEgrams and average PTS from three groups of chinchillas at least 30 days after being exposed to 1, 10, or 100 reverberant blast waves from Source I at 155 dB peak SPL. Each line represents the difference between the normative (n=102) mean DPEgram and the mean experimental group DPEgram. The open symbols represent the mean PTS for each group at the specified frequencies.

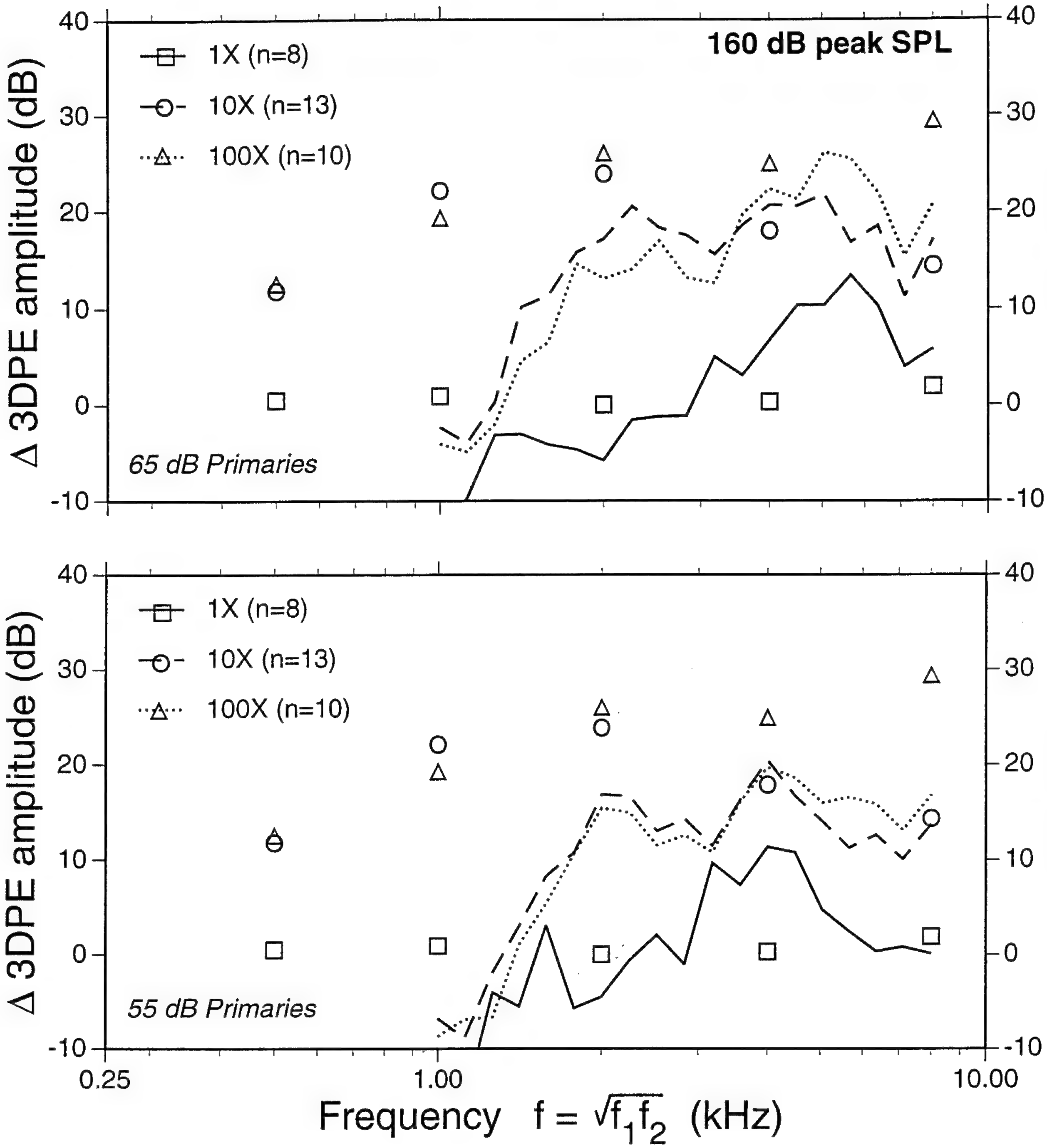

Figure 50. The group mean change in DPEgrams and average PTS from three groups of chinchillas at least 30 days after being exposed to 1, 10, or 100 reverberant blast waves from Source I at 160 dB peak SPL. Each line represents the difference between the normative (n=102) mean DPEgram and the mean experimental group DPEgram. The open symbols represent the mean PTS for each group at the specified frequencies.

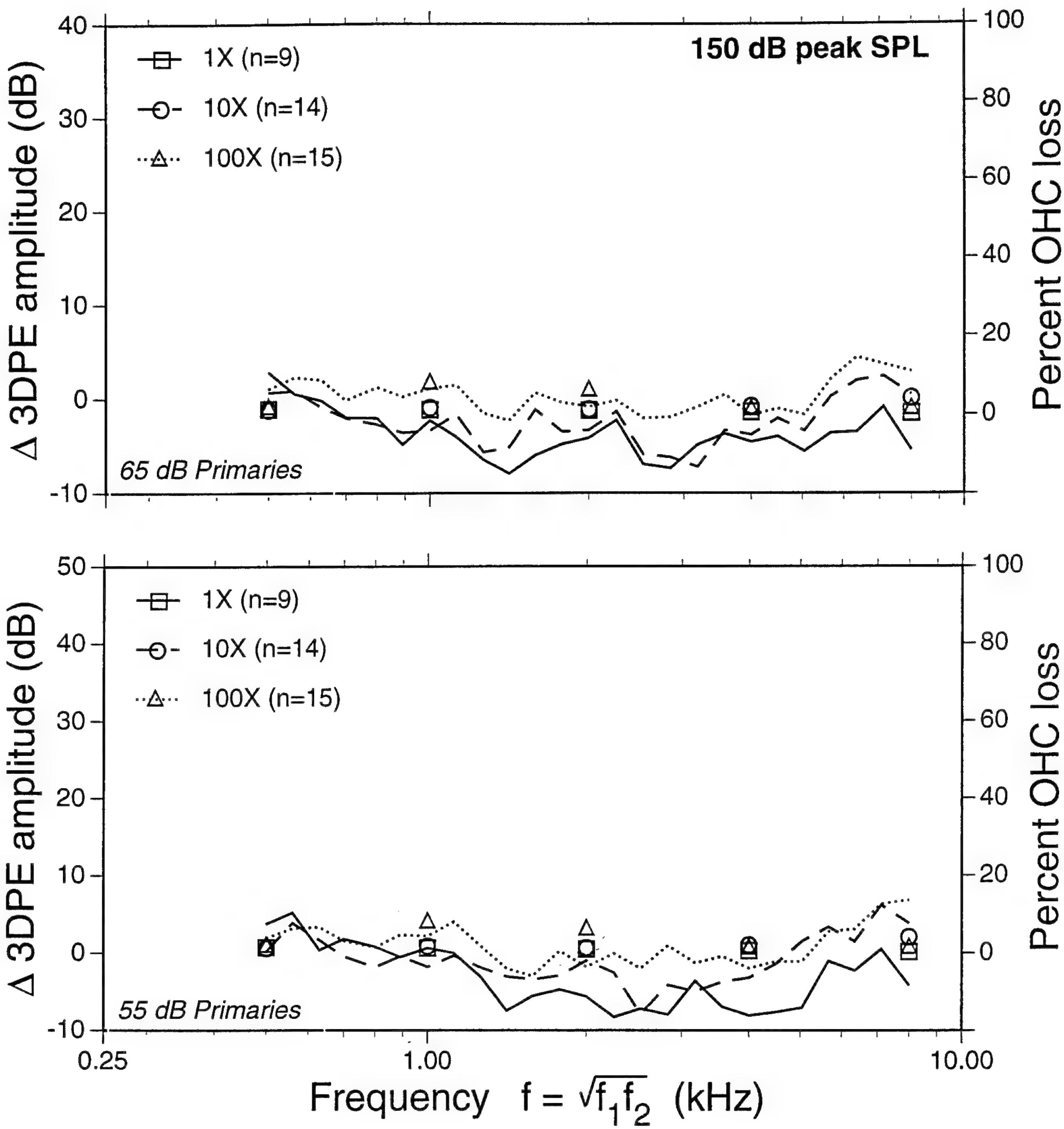

Figure 51. The group mean change in DPEgrams and average percent outer hair cell loss from three groups of chinchillas at least 30 days after being exposed to 1, 10, or 100 reverberant blast waves from Source I at 150 dB peak SPL. Each line represents the difference between the normative (n=102) mean DPEgram and the mean experimental group DPEgram. The open symbols represent the mean PTS for each group at the specified frequencies.

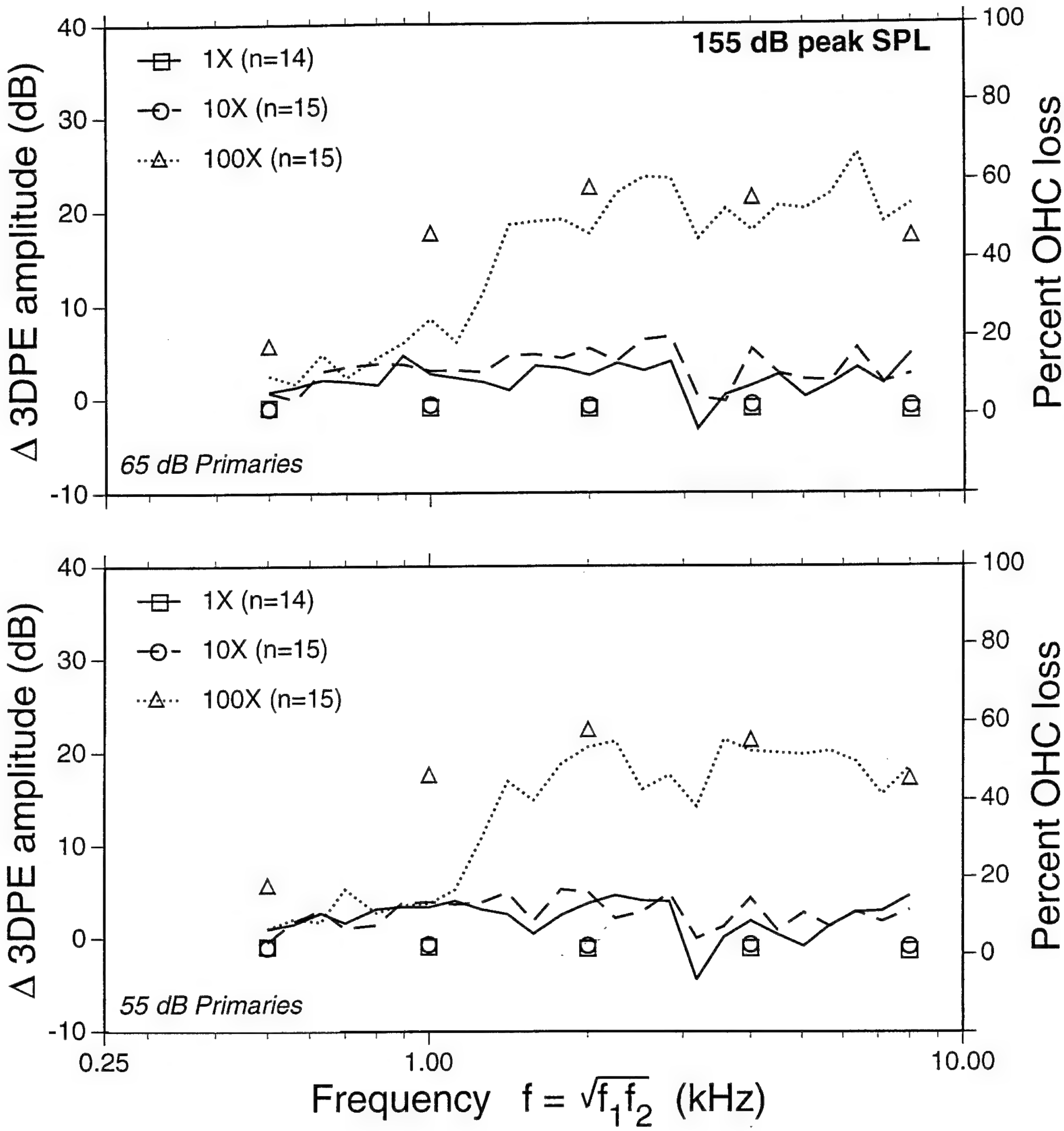

Figure 52. The group mean change in DPEgrams and average percent outer hair cell loss from three groups of chinchillas at least 30 days after being exposed to 1, 10, or 100 reverberant blast waves from Source I at 155 dB peak SPL. Each line represents the difference between the normative (n=102) mean DPEgram and the mean experimental group DPEgram. The open symbols represent the mean PTS for each group at the specified frequencies.

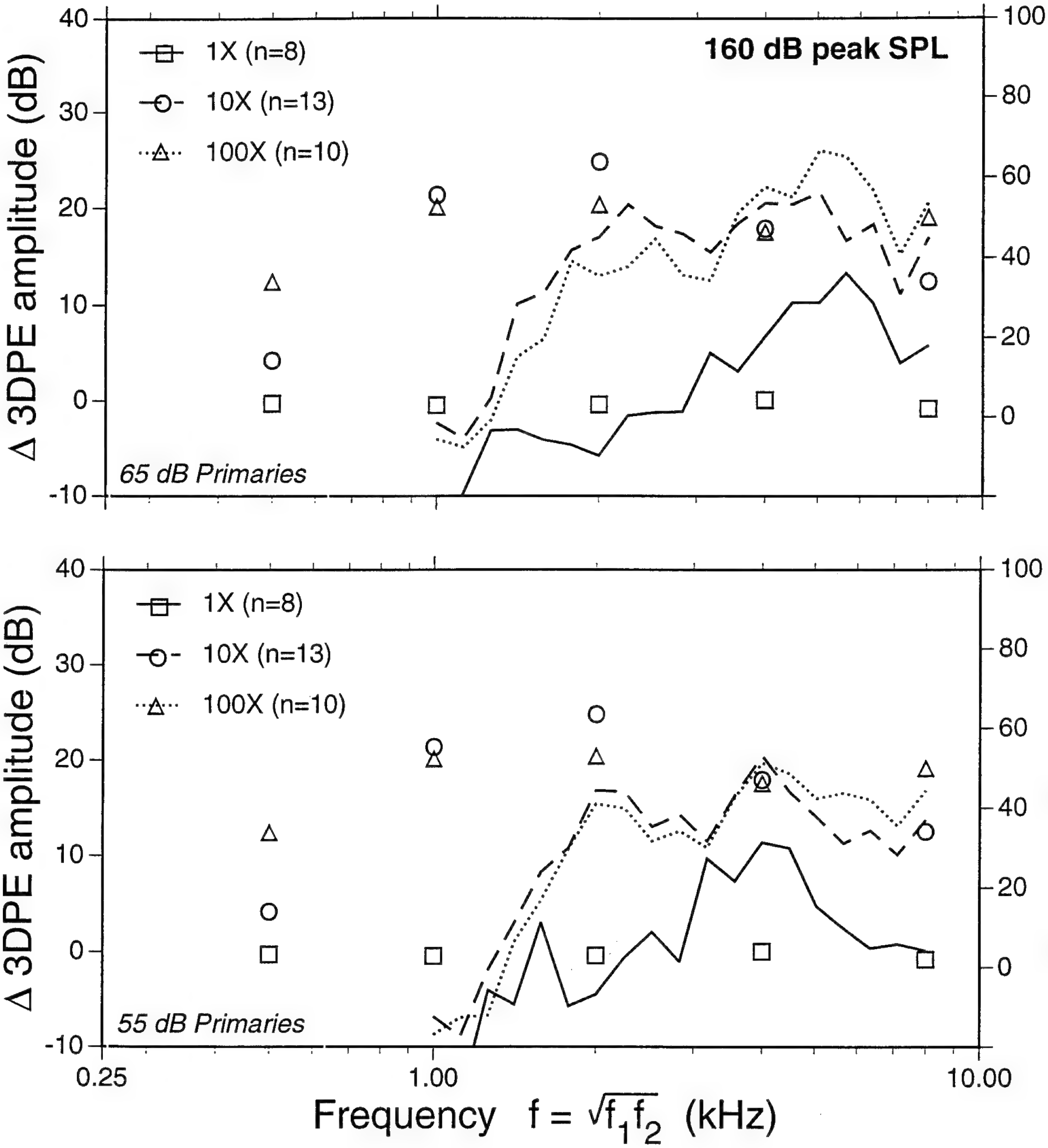

Figure 53. The group mean change in DPEgrams and average percent outer hair cell loss from three groups of chinchillas at least 30 days after being exposed to 1, 10, or 100 reverberant blast waves from Source I at 160 dB peak SPL. Each line represents the difference between the normative (n=102) mean DPEgram and the mean experimental group DPEgram. The open symbols represent the mean PTS for each group at the specified frequencies.

promulgated by the USDA, and will contribute to efforts to create an experimentally-based damage risk criterion.

Our data base on the auditory effects of blast wave exposure contains both the audiometric and histological results obtained from exposure of approximately 700 chinchillas to one of 102 different blast wave exposures. Parameters of the exposure were: exposure environment, either reverberant or nonreverberant; peak sound pressure level (150, 155, 160 dB); number of presentations (1, 10, 100); impulse presentation rate (1/min, 1/10 min, 10/min); and spectrum (four different sources have been used, each producing a blast wave having a different spectral distribution of energy[3]).

To be effective, a damage risk criterion (DRC) for exposure to blast waves must be reasonably easy to interpret and apply. An essential first step in development of a DRC is to establish metrics for quantifying the exposure and the trauma, and to demonstrate that the metrics chosen are highly correlated.

The fundamental question that this analysis should seek to answer is: How does threshold shift and sensory cell loss accumulate with an increasing exposure energy? Inherent in such a broadly stated question are the following four interrelated issues:

1. The energy of an exposure is increased by increasing the peak sound pressure level, the number of impulse presentations, and a change from a nonreverberant to a reverberant exposure environment. How is trauma[4] related to the exposure energy and how is this relation affected by the variables used to increase that energy?

2. While the total energy of an exposure stimulus can be the same for various sources, the distribution of energy across frequency can be very different for different blast wave sources. Different blast wave exposures are best compared on a spectrally-weighted energy basis in order to estimate trauma. What is the most appropriate spectral weighting function that should be applied to the spectrum of an impulse?

3. An issue of practical importance in the medical management of an acute acoustic trauma is the interpretation of early postexposure diagnostic audiometry. The suggested analyses will contribute to answering the question, how reliable a measure are the early postexposure threshold shifts in predicting the eventual permanent audiometric and histological changes?

4. Can "single number" variables such as $TS_{a,b,c}$, $PTS_{a,b,c}$ and OHC_T/IHC_T be used to provide an index of trauma, and are these variables highly correlated among themselves as well

[3] Note: The spectral distribution of energy from a given source is also altered by the exposure environment.

[4] Note: The use of the word trauma in a generic context refers to both audiometric and histological measures.

as with a "single number" metric used to describe the exposure.

Such an analysis would integrate all the data acquired on the previous DoD contracts mentioned earlier in a form that will provide answers to the issues raised in the above four points to the extent possible with this data base. Without such a comprehensive analysis, the results of this 12-year effort will exist as separate, somewhat disjointed reports from which it will be difficult at a later date to make generalizations that are applicable to human hearing conservation issues.

Military Significance and Relevance to USAMRDC mission needs. Exposure to high levels of impulsive noise are known to pose a high risk of permanent hearing loss. An acute hearing loss can manifest itself following a single exposure event or the hearing loss can be the result of chronic exposure (i.e. the result of the cumulative effect of exposure over several years of service). Severe exposures, even if only a large temporary threshold shift is incurred, can disable an individual to the extent that communicative abilities are severely compromised and the individual becomes a potential threat to himself and to others. Studies have shown, for example, that tank crews whose speech intelligibility (SI) is degraded from 100% to 75% suffer an increased mental work load while a SI decrease to 50% effects a target identification performance task (Whitaker et al., 1989). Severe acute hearing loss may also be a primary factor in the determination of casualty status for an individual.

Another important consideration is the economics of hearing loss. The costs of compensation and treatment for acoustic trauma are high and increasing at a rapid rate. Consider: (a) Estimates by Suter and von Gierke (1987) indicate that $125 million was paid out in compensation for hearing loss by the Veterans Administration in 1980. (b) In the 12-year period ending in 1980, this compensation amounted to approximately $1 billion. (c) Over the decade 1978-87 estimates for compensation for all Federal employees amount to $2.5 billion. In the military nearly 1 million employees are exposed to hazardous noise levels, much of it impulsive in nature. Thus, our ability to estimate or predict blast wave-induced hearing loss can be cost effective and can realize long-term savings in addition to preserving the quality of life for military personnel.

VI. ACKNOWLEDGMENTS

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Table 11. The analysis of variance summary tables for the audiometric (TS_{max}, PTS) and histological dependent (%OHC loss, %IHC loss) variables.

Analysis of Maximum Threshold Shift

Source of Variation	Sum of Squares	df	Mean Square	F	p(F)
Source (S)	54659.94	1	54659.94	106.23	.000
Peak (P)	51333.12	2	25666.56	49.88	.000
Number (N)	432704.66	2	216352.33	420.46	.000
S by P	8966.83	2	4483.41	8.71	.000
S by N	13186.23	2	6593.11	12.81	.000
P by N	20774.76	4	5193.69	10.09	.000
S by P by N	15223.41	4	3805.85	7.40	.000
Between Subjects	129155.89	251	514.57		
Frequency (F)	14907.59	2	7453.79	84.02	.000
S by F	24.51	2	12.26	.14	.871
P by F	1056.73	4	264.18	2.98	.019
N by F	4275.68	4	1068.92	12.05	.000
S by P by F	205.53	4	51.38	.58	.678
S by N by F	746.06	4	186.52	2.10	.079
P by N by F	2249.43	8	281.18	3.17	.002
S by P by N by F	891.38	8	111.42	1.26	.264
Within Subjects	44533.63	502	88.71		

Analysis of Permanent Threshold Shift

Source of Variation	Sum of Squares	df	Mean Square	F	p(F)
Source (S)	43053.57	1	43053.57	40.05	.000
Peak (P)	39161.79	2	19580.90	18.21	.000
Number (N)	191174.33	2	95587.16	88.91	.000
S by P	63.37	2	31.69	.03	.971
S by N	30686.23	2	15343.11	14.27	.000
P by N	24992.59	4	6248.15	5.81	.000
S by P by N	3793.74	4	948.44	.88	.475
Between Subjects	265539.45	247	1075.06		
Frequency (F)	8748.35	6	1458.06	21.64	.000
S by F	953.21	6	158.87	2.36	.029
P by F	2312.39	12	192.70	2.86	.001
N by F	6165.06	12	513.76	7.63	.000
S by P by F	2209.78	12	184.15	2.73	.001
S by N by F	860.25	12	71.69	1.06	.387
P by N by F	4746.17	24	197.76	2.94	.000
S by P by N by F	2685.00	24	111.87	1.66	.024
Within Subjects	99849.03	1482	67.37		

Table 11 (continued). The analysis of variance summary tables for the audiometric (TS_{max}, PTS) and histological dependent (%OHC loss, %IHC loss) variables.

Analysis of Percent Outer Hair Cell Loss

Source of Variation	Sum of Squares	df	Mean Square	F	p(F)
Source (S)	111936.87	1	111936.87	41.76	.000
Peak (P)	118891.74	2	59445.87	22.18	.000
Number (N)	478544.84	2	239272.42	89.27	.000
S by P	6612.53	2	3306.26	1.23	.293
S by N	69856.21	2	34928.11	13.03	.000
P by N	71348.43	4	17837.11	6.65	.000
S by P by N	14335.51	4	3583.88	1.34	.257
Between Subjects	678130.88	253	2680.36		
Frequency (F)	212261.17	7	30323.02	114.49	.000
S by F	47257.65	7	6751.09	25.49	.000
P by F	24556.49	14	1754.04	6.62	.000
N by F	147164.16	14	10511.73	39.69	.000
S by P by F	13209.14	14	943.51	3.56	.000
S by N by F	25350.98	14	1810.78	6.84	.000
P by N by F	38283.26	28	1367.26	5.16	.000
S by P by N by F	23433.65	28	836.92	3.16	.000
Within Subjects	469069.46	1771	264.86		

Analysis of Percent Inner Hair Cell Loss

Source of Variation	Sum of Squares	df	Mean Square	F	p(F)
Source (S)	12340.43	1	12340.43	23.56	.000
Peak (P)	17517.85	2	8758.92	16.73	.000
Number (N)	41998.56	2	20999.28	40.10	.000
S by P	1315.36	2	657.68	1.26	.287
S by N	10721.11	2	5360.55	10.24	.000
P by N	8810.00	4	2202.50	4.21	.003
S by P by N	806.41	4	201.60	.38	.819
Between Subjects	132492.79	253	523.69		
Frequency (F)	25715.29	7	3673.61	29.95	.000
S by F	12474.80	7	1782.11	14.53	.000
P by F	8210.99	14	586.50	4.78	.000
N by F	24857.28	14	1775.52	14.48	.000
S by P by F	2808.56	14	200.61	1.64	.063
S by N by F	8828.66	14	630.62	5.14	.000
P by N by F	9164.07	28	327.29	2.67	.000
S by P by N by F	6537.57	28	233.48	1.90	.003
Within Subjects	217214.16	1771	122.65		

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